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THE MORTALITY OF APPENDICITIS.[1]

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THE subject of this address was chosen because there has been much talk of appendicitis, and my house surgeon (Dr. Enid Anderson) had indexed the patients in my practice at the Royal Prince Alfred Hospital. I propose to analyse quickly the death rate at the hospital as a whole and then to give some figures from the Government Statistician's office. These will be followed by an examination of the results of treatment of my own patients. Then I intend to mention some points of diagnosis, technique and treatment. I shall account to you for a dozen fatalities.

The figures here given have been altered slightly since their presentation at Newcastle. It was discovered after the address was delivered that a certain number of patients shown in the records as "remaining in hospital" on the last day of each year is carried forward into the following year's figures. The total number is increased, and unless the gross number "remaining in hospital" be subtracted from the total of patients, the death rate is artificially made less.

In my series another fatal case of "appendicitis with abscess" has been added. The notes of this patient are not shown in the records under "appendicitis" and have been filed evidently under "ventral hernia". They concern an old lady of sixty who had

[1] The substance of this paper was read at a meeting of the Central Northern Medical Association, at Newcastle, New South Wales, on March 20, 1935. Certain alterations and additions have since been made.

had a large hernia for years, with intermittent attacks of pain, thought to have been due to incarceration, but which had been caused by attacks of appendicitis in the sac. The case was reported in a paper on "Acute Abdominal Conditions" in THE MEDICAL JOURNAL OF AUSTRALIA of April 2, 1932.[1] This death has been added to the hospital figures also.

Let us take Tables I and Ia. These are the figures for all surgeons of the hospital. It was apparent that for some reason there had been a sudden improvement of the results in 1926 of the second type ("Appendicitis with Abscess"). The figures have been divided into two periods: (i) 1920-1925 (six years), and (ii) 1926-1934 (nine years).

TABLE I.

Cases from the Royal Prince Alfred Hospital.

Year.	Appendicitis. Acute and Chronic.		Appendicitis with Abscess.		Appendicitis: Appendix Ruptured with General Peritonitis.	
	Patients.	Deaths.	Patients.	Deaths.	Patients.	Deaths.
1920 ..	352	5	46	5	11	5
1921 ..	426	3	56	8	6	2
1922 ..	450	3	41	5	9	3
1923 ..	515	3	22	2	6	4
1924 ..	537	1	34	4	7	5
1925 ..	552	0	34	5	8	3
1926 ..	665	0	31	1	10	4
1927 ..	732	1	36	0	22	8
1928 ..	751	1	43	0	18	9
1929 ..	632	2	26	1	11	6
1930 ..	497	3	33	1	9	1
1931 ..	439	3	19	0	10	5
1932 ..	317	5	25	0	9	0
1933 ..	338	3	15	1	9	5
1934 ..	656	3	25	0	14	5
Total ..	7,859	36	486	33	159	65
Remaining in hospital	309		26	+1	7	
Final total	7,550	36	461	34	152	65

It will be noticed in the case of appendicitis, acute and chronic, that the death rate has fallen from 0·55% to 0·43%. In the second type there has been an almost incredible reduction from 13·18% to 2·07%.

A member of the Statistician's Department said that the departmental officers would not believe such a change without checking. So I checked the figures carefully and found them correct. They are taken from the annual reports. I should like to thank Dr. Herbert Schlink, who introduced a good method of recording in 1910, for making it possible to give you these conclusions.

In the third type ("Appendicitis: Ruptured Appendix with General Peritonitis") there has been a slight improvement of the dire results from 48·88% to 40·18%.

There are many possible factors in the betterment of the issue of each type. I think the main factor is that we no longer greet the patient with one hand and thrust a knife into his abdomen with the other. We have become more conservative with certain conditions.

The use of the electric suction apparatus obviates spilling of infective fluids into the peritoneal cavity. Gas gangrene antiserum seems to be useful, as does the intravenous injection of 10% glucose in normal saline solution. (The glucose should be specially prepared by a reputable firm and sealed in pure glass.)

Since the improvement of the roads and ambulances the patients have had an easier ride to hospital.

It appeared, on reading the histories, that to be classified in the third type the patient had to have already two feet in the grave.

It is my intention to suggest to the authorities a different method of classification according to the condition of the appendix. This question will be discussed later.

TABLE Ia.

Period.	Appendicitis, Acute and Chronic.			Appendicitis with Abscess.			Appendicitis: Appendix Ruptured with General Peritonitis.		
	Patients.	Deaths.	Rate.	Patients.	Deaths.	Rate.	Patients.	Deaths.	Rate.
First period, 1920 to 1925 (six years)	2,707	15	% 0·55	220	29	% 13·18	45	22	% 48·88
All types: 2,972 patients, 66 deaths, rate 2·22%.									
Second period, 1926 to 1934 (nine years)	4,843	21	0·43	241	5	2·07	107	43	40·18
All types: 5,191 patients, 69 deaths, rate 1·32%.									
Total years, 1920 to 1934 (fifteen years).. ..	7,550	36	0·47	461	34	7·37	152	65	42·76
All types: 8,163 patients, 135 deaths, rate 1·64%.									

Death rate (all causes) in New South Wales: 9 per 1,000 (approx.) of mean population.
Death rate (appendicitis, acute and chronic), Royal Prince Alfred Hospital: 4·7 per 1,000 of case incidence.

You will note that the death rate from all causes in New South Wales is roughly 9 per 1,000 inhabitants. It varies little on either side of nine.

Now the death rate of "appendicitis, acute and chronic", at the Royal Prince Alfred Hospital is 4·7 per 1,000, so that you have the anomaly that if you are in Prince Alfred Hospital with appendicitis (Type I) you have a two to one better chance of living than the man in the street. Of course, this is a statistical joke; the patient is in hospital only for about two weeks and obviously has less chance of dying in a fortnight than he has in fifty-two weeks. Also the statistics refer to people of all ages.

Now in regard to Tables II and IIA. You will see that the death rate in New South Wales for appendicitis is remarkably constant, the highest being 0·89 per 10,000 in 1923, and the lowest 0·70 in 1924.

TABLE II.

Deaths in the State of New South Wales.

Year.	Appendicitis.	Rate per 10,000 Mean Population.	Accident.	Rate.	Due to Motor Cars.
1920 ..	165	0·80	937	4·98	Not obtained.
1921 ..	155	0·74	980	4·65	Not obtained.
1922 ..	177	0·82	953	4·42	Not obtained.
1923 ..	197	0·89	964	4·38	111
1924 ..	156	0·70	1,079	4·81	127
1925 ..	178	0·78	1,141	4·97	227
1926 ..	187	0·80	1,264	5·39	256
1927 ..	201	0·84	1,425	5·93	335
1928 ..	190	0·77	1,314	5·34	384
1929 ..	216	0·86	1,455	5·81	478
1930 ..	211	0·83	1,248	4·93	431
1931 ..	197	0·77	1,071	4·19	332
1932 ..	214	0·83	1,100	4·26	304
1933 ..	212	0·81	1,067	4·10	321
Total..	2,656 (14 years)		15,998 (14 years)		3,306 (11 years)

NOTE.—From 1927 to 1930 open verdicts were included with Accidents.

TABLE IIA.

Deaths from Appendicitis for one Year (1933) in New South Wales.

Place.	Number.
General hospitals	149[1]
Private hospitals	53
Military hospitals	1
State homes	1
Not in hospital	8
Total	212

[1] In Royal Prince Alfred Hospital, 9 deaths.

It was decided to compare the lethal effect of appendicitis with that of accident. You observe how the rate of violent death increases in prosperous times, also that all accidents, or even motor car accidents alone, are much more serious for the community than is appendicitis. In 1933 there were 212 deaths from appendicitis and 321 from motor accidents. The morbid craze for speed is without question the chief factor in the high death rate of motor accidents. It is high time that people ceased to rush as if their life depended on reaching their destination a few seconds before the other fellow. Life is too short and occasionally too precious for all this unjustifiable bustle.

You will note that of 149 deaths from appendicitis in general hospitals for the year 1933, nine occurred in the Royal Prince Alfred Hospital. (The term "general hospital" includes all hospitals under the Hospitals Commission, and on June 30 there were 174. Many of these are small hospitals and are negligible in considering the question.)

If you will turn to Tables I and IA and look at the figures for 1932, it will be noticed that there were five deaths from "appendicitis, acute and chronic", but no deaths from the more serious conditions. So that if one were to consider this particular year, the erroneous opinion might be formed that a perforated appendix with peritonitis was less harmful than ordinary appendicitis. This is mentioned to show how fallacious it can be to draw conclusions from a very small series.

To be accurate one would have to read each history in order to determine the condition found at operation, the number of readmissions, and so on, but I had neither the time nor the inclination to wade through over eight thousand records of the period in question. So it must be understood that the figures refer to patients whose disease has been filed in the records by various registrars as "appendicitis".

Now turn to Tables III to IIIE, which are the records of my own patients at the Royal Prince Alfred Hospital from April, 1920, to June 30, 1934 (fourteen years and three months). The numbers do not include such patients as were admitted under my care but treated by a colleague, nor the patients who were operated on by a resident medical officer under my supervision.

TABLE III.

Author's Patients at Royal Prince Alfred Hospital, 1920 to 1934 (14 years 3 months).

Total, 925. Males, 553. Females, 372.
Youngest, 4 years 6 months. Oldest, 81 years.

Decade.	Number.	Deaths.	Percentage.
First	19	0	—
Second	345	3	0·87
Third	283	0	—
Fourth	126	2	1·58
Fifth	90	0	—
Sixth	40	1	2·5
Seventh	19	5	26·31
Eighth	2	1	50·00
Ninth	1	0	—
Age 4 to 49	863	5	0·58
Age 50 to 81	62	7	11·29
Total	925	12	1·29

TABLE IIIA.

Type.	Number.	Deaths.	Rate.
			%
Acute and chronic	844	2	0·23
With abscess	65	4	6·15
Ruptured appendix with general peritonitis	16	6	37·5
Total	925	12	1·29

TABLE IIIB.
Type of Operation.

Operations.	Number.
Appendicectomy	761
Appendicectomy with drainage	103
Drainage only	31
Laparotomy	2
Resection	1
Total	898

TABLE IIIC.
Condition of Appendix.

Condition of Appendix.	Number.	Deaths.	Rate.
			%
Gangrenous	148	9	6·08
Acute inflammation	347	1	0·28
Subacute inflammation	201	0	
Chronic inflammation	124	0	
Abscess	26	2	7·69
Appendicectomy after abscess	14	0	
Normal	20	0	
Indeterminable, including no operation	45	0	
Total	925	12	

TABLE IIID.
Operation Mortality in All Types.

Number of Operations.	Deaths.	Rate.
		%
898	11	1·22

These figures are open to the fair criticism that some cases may have been missed in searching the records, owing to the possibility of human error. Knowing the searchers, I do not think that this mistake is a very present one.

You will note that the youngest patient was aged four years and six months, and the oldest eighty-one years. The very youngest children go to the Royal Alexandra Hospital for Children, yet there were nineteen patients in the first decade of life. I understand that appendicitis in young babies is very insidious and that many cases are diagnosed *post mortem*.

The patient aged eighty-one is Number 677 of "Appendicitis with Abscess" series. The notes show that he had free pus and that the abdomen was drained without removal of the appendix. He recovered.

It will be noticed that the age of the patient is an important factor in the prognosis. At the time of life when the disease is commonest, the death rate is, luckily, the lowest. You will see that from the age of four to forty-nine years there were 863 cases with five deaths (rate 0·58%), whilst from the age of fifty to eighty-one years there were 62 cases and seven deaths (rate 11·29%).

Diagnosis is more difficult in the aged and resistance is less. The condition of the appendix is, of course, a most important consideration. When gangrene of any part of the appendix was present the death rate was 6·08%. With acute inflammation there was only one death in 347 cases (rate 0·28%). There were no fatal cases where the inflammation was described as subacute or chronic, nor in the 20 patients whose appendix was recorded as normal.

There were two deaths of patients whose abscess was drained without appendicectomy (Numbers 5 and 12 of my own fatal cases). No criticism of the procedure of simple drainage could be made in either of these cases.

The method is so safe that when one patient, who had been treated by drainage of what was apparently an appendiceal abscess, died, one said at once that he might have had another condition, and at the *post mortem* examination it was found that the abscess had been caused by a leaking carcinoma of the ascending colon.

I was surprised to find that I had employed drainage as often as 103 times as an addition to appendicectomy. I knew that I had not hesitated to use a drainage tube when it was indicated, but I did not think that it had been used so often.

By drainage I do not mean that a large tube is put into the abdominal cavity and left for several days or a week. A soft tube is always used and is removed in twenty-four, forty-eight or seventy-two hours. It is rarely left longer and can do nothing but harm if allowed to remain.

Other factors of importance in causing mortality are the duration of the illness before the patient's admission to hospital and the taking of purgatives. I do not think that the awful results of the third type can be improved until the patients report earlier to their doctors and refrain from taking a purge when an unusual pain is felt in the abdomen.

Most medical men appreciate the danger of a purge, but many do not realize that an enema can be fraught with greater harm. I once mopped from the peritoneal cavity of a soldier the contents of three enemata which had reached the inside of the abdomen by way of a perforation of the appendix. I have seen also a diverticular abscess burst apparently by an enema. You see in the operation records one case of resection. This concerns a

TABLE IIIE.
Duration of Illness before Patient's Admission to Hospital.

Type of Inflammation.	Days.						Several Days.	Weeks.	Not Specified.	Total.
	½	1	2	3	4	5 or more.				
Acute	40	102	61	30	14	41	6	28	25	347
Gangrenous	14	32	41	15	16	14	4	5	9	148

patient in whom I found the appendix and caecum hard and indurated, but freely mobile. I did not know whether I was dealing with neoplasm, tuberculous inflammation or chronic induration of ordinary origin. The condition was obviously operable, so I resected the caecum and the patient did very well. The pathologist reported inflammation with no sign of tuberculosis. There is a condition in which the whole caecum becomes hard and woody and glued down. It is wise to refrain from all interference when one meets such a state of affairs.

With regard to classification, the present hospital method depends on the mental vagaries of each registrar. My fatal case Number 1 is filed under "acute appendicitis"; so, too, is the record of a patient whose appendix is described in the notes as "normal".

Peritonitis is seldom general in the sense that every square inch of the serous membrane is affected, and all gangrenous cases have some peritonitis, even when there has been no rupture.

It would be better to classify cases according to the condition of the appendix. There should be a heading "normal appendix". If one removes a normal appendix, it should be charged to one's account, and if a surgeon were to remove an excessive number of normal appendices, somebody should ask pertinent questions. It may be granted that an appendix can be apparently normal and yet have been the cause of previous trouble.

When I look at all these figures I am reminded of two very dear Irish gentlemen whom I knew. One was a surgeon and the other, from the nature of his work, never used a knife. The former was relating the number of a certain type of operations he had performed. The second man just looked over his glasses and asked: "Were they all necessary?"

You could ask me the same question and my frank reply would be: "No!" The patients who come under my care have been seen by one or more colleagues and have been dubbed appendicitis, and it takes a good deal of intestinal fortitude to say that a given patient has not appendicitis when several other men have announced that he has. However, one refuses to operate in some cases, and one should take reasonable care to operate only when one honestly thinks that it is in the best interests of the patient to do so.

Now I ask you to regard Tables IV and IVA. This morning I had some time to spare and looked up the records of the ten-year period 1910-1919 and got some surprises. In the first type the mortality rate has shown a steady decrease in the three periods, from 0·61% to 0·55% to 0·43%. In the second type there is a remarkable fluctuation from 6·32% to 13·18% to 2·07%. In the third type there has been no improvement and the rates are 38·68% to 48·88% to 40·18%. Fortunately there have been only 289

TABLE IV.

Royal Prince Alfred Hospital, 1910 to 1919 (ten years).

Year.	Appendicitis. Acute and Chronic.		Appendicitis with Abscess.		Appendicitis: Appendix Ruptured with General Peritonitis.	
	Patients.	Deaths.	Patients.	Deaths.	Patients.	Deaths.
1910 ..	328	0	40	2	15	4
1911 ..	297	4	53	3	20	7
1912 ..	356	1	70	4	27	7
1913 ..	361	3	67	3	26	5
1914 ..	395	1	50	0	12	9
1915 ..	377	1	30	4	5	4
1916 ..	344	3	35	0	8	2
1917[1] ..	—	—	—	—	—	—
1918 ..	482	2	49	4	14	5
1919 ..	284	4	50	6	18	10
Total ..	3,224	19	444	26	145	53
Remaining in hospital	144		33		8	
Final total[2]	3,080		411		137	

[1] No annual report. Period changed from December 31 to June 30.
[2] The rates in the above groups are: 0·61%, 6·32%, 38·68%.

TABLE IVA.

Author's Patients at Royal Prince Alfred Hospital.

Period.	Appendicitis, Acute and Chronic.			Appendicitis with Abscess.			Appendicitis: Appendix Ruptured with General Peritonitis.		
	Patients.	Deaths.	Rate.	Patients.	Deaths.	Rate.	Patients.	Deaths.	Rate.
First period, 1920 to 1925 (5 years and 3 months)..	305	1	% 0·32	25	3	% 12·0	1	1	% —
All types: 331 patients, 5 deaths, rate 1·51%.									
Second period, 1926 to 1934 (nine years).. ..	539	1	0·18	40	1	2·5	15	5	33·33
All types: 594 patients, 7 deaths, rate 1·17%.									
All Surgeons.									
Total all years, 1910 to 1934 (twenty-five years)..	10,630	55	0·51	871	60	6·88	289	118	40·83
Grand total: 11,791 patients, 233 deaths, rate 1·97%.									

such patients in a total of 11,791 in twenty-five years.

I wondered why the rate should have increased in the period 1920-1925, and the first thing that occurred to one's mind was that with three others I was appointed to the hospital in 1920.

I looked up the records to find the results of the cases treated by the older members of the staff and compared them with the issue when the new appointees were at the helm. The figures are:

Type 2.—"Appendicitis with abscess."
Senior: 114 patients, 13 deaths (rate 11·40%).
Junior: 106 patients, 16 deaths (rate 15·09%).

Type 3.—"Appendicitis: Ruptured appendix with general peritonitis."
Senior: 24 patients, 16 deaths (rate 66·66%). (One case under the care of a physician.)
Junior: 21 patients, 6 deaths (rate 28·5%).

So that I think it will be admitted that the acquisition of four new surgeons was not a serious factor in putting up the mortality rate from 1920 to 1925. It will be noted that the grand total for twenty-five years of all types of the disease is 11,791 patients, with 233 deaths, or a mortality rate of 1·97%. Although my own series is relatively small, I thought it would be wise to divide it into two periods, April, 1920, to June, 1925 (five years and three months), and July, 1925 to June, 1934 (nine years), for comparison with hospital figures of similar periods. The results are shown in the table. The second period shows improvement on the first.

I should like to mention one or two interesting cases.

In Number 633 (Type 2) I was astonished to find the operation note: "Draining abscess, appendicectomy, radical cure of right inguinal hernia." My mind was relieved to find that the record was of a female patient, aged seventy-seven years, who had been ill for some months and who had had a hernia for years. I had diagnosed incarceration of a right inguinal hernia, but found that she had an appendiceal abscess in a right hernial sac. The resident medical officer flattered me when he noted a radical cure of the hernia. The patient recovered in spite of everything.

In one case the appendix is described as having been eight inches long, no mention being made of a measurement. In another, the organ is said to have been seven inches long and half an inch in diameter.

Number 4,061 was a male, aged thirty years, who had been ill for some days and who was thought to have a perforation of a duodenal ulcer.

Number 3,827 is described as having an appendix "slightly inflamed", but a double salpingectomy was performed. There must be some doubt whether appendicitis was the real cause of the illness.

In Number 4,227 the appendix was "acutely inflamed" and a right salpingectomy was performed. Probably in this patient the tubal inflammation was secondary to appendicitis.

Number 6,540 is described as being a case of "woody phlegmon of the caecum". The appendix was not removed.

Number 6,709 was a patient who had a post-operative pulmonary embolism but recovered.

In nine patients the appendix is reported to have contained threadworms. No doubt in some of these cases administration of a worm powder would have been wiser treatment than appendicectomy, but in others the parasites had set up definite inflammation.

In one case a grape seed was found in the appendix, and in another a melon seed. Contrary to popular opinion, foreign bodies are found but rarely.

Short notes of all the fatal cases are appended.

In regard to the first, I mention that the pathologist was a resident medical officer, not out of disrespect, but to show that he was inexperienced. There is no doubt whatever that it was not the purse string that was at fault, but the condition of the caecum. Possibly I should have performed caecostomy.

Number 2 is remarkable in that until I had read the notes I had no memory whatever of having lost a patient suffering from appendicitis upon the operating table at the Royal Prince Alfred Hospital.

The "foul breath" noted in Number 3 is of a distinctive odour and is usually called the "appendiceal breath". It would probably have been wiser to have refrained from performing appendicectomy and to have been content with drainage.

Number 5 is a sad case and I hope that I shall not meet such another. I think of this patient often when operating for appendicitis and am careful about hæmorrhage. Nevertheless, I thought that I was being extra careful with this patient. Being a female, she could not have been a true "bleeder", but must have had some hæmorrhagic diathesis. You note that I did not divide any structure within the abdomen.

Number 6 was inoperable, although the condition was not diagnosed till the *post mortem* examination was made.

In Number 7 the patient, so far as I know, provides the only instance in which jejunostomy was performed.

Patient Number 8 was treated by me for the inflammation of the liver and not for appendicitis.

In Number 9 you will note that there was "free pus" and that the appendix was gangrenous. Yet five days after the operation the patient died and the pathologist found the "peritoneum normal" and "no fluid nor pus". The patient died of nephritis.

Number 12 is not in the records under "appendicitis", but has been included because I remember the patient. The case is worth remembering, for things are not always what they seem.

You will observe that six of the above patients were over sixty years of age and that a seventh was fifty-eight. In five cases a note is made that the patient had taken a purgative. The remainder may or may not have been so foolish. Patient Number 9 came from Helensburgh (28 miles). A journey, especially a rough one, may have a very baneful effect.

When a man puffs out his chest and announces or writes that he has treated so many hundreds of

cases of appendicitis and has never had a death, you should treat him as Captain Corcoran was treated in Gilbert and Sullivan's "H.M.S. Pinafore". You will remember that the captain sings "Tho' 'bother it' I may occasionally say, I never use a big big 'D' ". To this the crew replies: "What, never?" and the skipper: "Hardly ever". Or you can treat him as a poker player and say: "I'll see you; show your cards"—in other words, "produce the records". As a colleague said the other day, there are two words which should be banished from medical literature: one is "never" and the other "always". They should be replaced by "hardly ever" and "generally".

With regard to diagnosis, this is quite easy in a typical case, but all patients do not "play fair". It is the unusual cases which cause difficulty. I need not remind you that the initial pain is due to tension in the appendix and is referred to the branches of corresponding intercostal nerves. It is usually felt near the umbilicus, but may be experienced anywhere in their distribution. The local pain is due to irritation of the extraperitoneal nerve endings and is felt as tenderness typically in the right iliac fossa. It may be demonstrated by rectal examination when the appendix is in the pelvis. On the other hand, the local manifestation may be absent when the organ is deeply situated. If you encounter a young male adult who has something wrong in the abdomen, and if you merely guess at appendicitis, your opinion is likely to be right. Remember that if you see a little bird on the eaves of your house, it may be a canary, but is more likely to be a sparrow.

What guides one most in the difficult cases is the "general cut" of the patient and, what is more, one cannot describe the reason of one's opinion. The "appendiceal breath" is often a great help. The theoretical cutaneous hyperæsthesia is of no practical use.

A leucocyte count is not of much help in guiding one whether to operate or not. Nor will it distinguish between appendicitis and pneumonia, for example. It may be of use in differentiating between appendicitis and typhoid fever, but even here I would rather trust my nose.

The rotation test of Zachary Cope may give a positive result, but is not of as much value as the books lead one to believe.

The pain felt in the caecal region when pressure is applied abruptly, but gently, in the left subcostal region has been observed by us all for years and has had some surgeon's name tacked on to it lately.

With regard to operation, it can be agreed that when the patient is seen early in the attack it is wise to operate. However, when the illness has been present for some days—one cannot define a time limit—and the patient is beating the infection, looking, eating and sleeping well, it is just as wise to defer active treatment till some time after the attack, whether there be a tumour or not and whatever the leucocyte count may be. A very high temperature is rare and generally excludes a diagnosis of appendicitis (except in children). A slow pulse is often accompanied by a serious intraabdominal condition, and a quick pulse is easily excited in nervous patients. However, a rising pulse rate is generally of serious import.

Now a few remarks on technique. I prefer the lateral muscle-splitting incision. If the case be acute, it is wise to make the skin incision in line with the fibres of the external oblique muscle, and it can be extended up or down as required. There is a little vessel running across the junction of the aponeuroses. This sometimes gives trouble and is best avoided. Keep away from small intestine. I am persuaded that a mid-line or lateral rectus incision makes the case more serious.

In cases of frank walled-off abscess simple drainage is sound and safe. The site of drainage depends on the situation of the appendix. If the organ be lateral in position, a stab drain in the loin is satisfactory. If the trouble be central, I generally drain through the wound. In the case of patients who have an acutely inflamed appendix which is removed in the presence of spilled pus or *débris*, drainage for forty-eight hours can do but good.

For anæsthesia I prefer ether. I know by experience that ethyl chloride followed by ether is a dangerous sequence and simply will not allow its use. When the inflammation has been acute, the wound should be sewn up with wide gaps between sutures, and it is wise to put a small drain down to the peritoneum.

Silk is usually used to ligate the appendix, and fine silk for the purse string which is generally employed. This was taught by Sir Alexander MacCormick, whose assistant I had the honour to be and whom I would like to thank for most of the little that I know. I can hear him now, as he ties the silk round the appendix, saying: "That will be there for some days." When the appendix is very thick, it is wise to cuff the base by "ring-barking" the peritoneum about one inch from the caecal junction. If the tip cannot be delivered easily, the appendix is removed base first.

In after-treatment, the less fuss, the better. Do not whip tired intestines, but wait for them to recover their tone. Vomiting is treated by gastric lavage and, if persistent, it is good treatment to give intravenous injections of glucose (with all precaution). Let the patient lie in bed as he likes, but encourage him to lie towards the right. I do not think that Fowler's position is as important as is maintained.

It takes a team of horses to make me reopen the abdomen. Such a step is occasionally necessary, but generally has a sad ending.

Now I have done, but I would express the wish that we all should have the humility of wisdom rather than the pride of knowledge. It would be good for us all to have a little card placed for daily observation with Cowper's beautiful lines typed boldly upon it:

Knowledge and Wisdom, far from being one,
Have oft-times no connexion. Knowledge dwells
In heads replete with thoughts of other men;
Wisdom in minds attentive to their own.

Knowledge is proud that he has learn'd so much;
Wisdom is humble that he knows no more.

Acknowledgement.

I wish to thank my house surgeons, Dr. Enid Anderson and Dr. Jobson, and many of their colleagues for their help in searching the records.

Appendix.

The following are the case histories of the patients in my own series who died.

CASE I.—Number 3,382, a male, aged sixteen years, is classified under the group "acute and chronic appendicitis". He was admitted to hospital on May 24, 1920, having been ill for two days and having taken salts each day. Appendicectomy with drainage was performed on the day of admission. The appendix was found to be gangrenous from its tip to the caecum, and there was free fluid and pus. The patient died on May 30 and *post mortem* examination through the wound, performed by the resident pathologist, revealed that the "purse string had torn the soft inflamed gut wall" and that there was gas in the peritoneal cavity.

This fatality is exactly analogous to the deaths which occur after anastomosis of the large bowel of which the blood supply has been damaged. Such patients do well for some days and die fairly suddenly on the third to fifth day. If one meets a condition of sloughing caecum, it is wise to perform caecostomy. I do not remember the condition of the caecum at the operation. The purse string was not the cause of the trouble.

CASE II.—Number 167, a male, aged fifty-eight years, is classified under the group "ruptured appendix with general peritonitis". He was admitted to hospital on February 15, 1923, and was operated upon on that day. He had been ill for eight days and had taken castor oil. His appendix was acutely inflamed and ruptured and there was general peritonitis. The appendix had been removed when the patient suddenly ceased breathing and died. The case was reported to the coroner.

CASE III.—Number 187, a male, aged thirty-three years, is classified under the group "ruptured appendix with general peritonitis". He was admitted to hospital on March 2, 1926, and was operated upon that same day. The patient had been ill for two days. The pain was not localized, the breath was foul and the breathing entirely thoracic. At operation free fluid was found, fæcal-like in appearance and odour. The patient showed signs of collapse, a tube was put in and the peritoneum was sewn up. The patient's condition improved and it was decided to remove the appendix. A ruptured gangrenous appendix was found and was removed with drainage. On March 3 the patient's condition was much improved, on March 4 he was not so well, and on March 5 he was much worse. The bowels were kept open with enemata. The patient vomited and 500 cubic centimetres of a 10% solution of glucose were administered intravenously; insulin was also given. At 12 o'clock midnight the patient's condition had improved. The improvement continued on March 6, 1926, and the stomach was again washed out. Further glucose was administered. On March 7, 1926, at 1.30 a.m., the patient died.

CASE IV.—Number 201, a male, aged sixty-two years, is classified in the group "ruptured appendix with general peritonitis". He was admitted to hospital on November 9, 1926. He had been ill for two days and had taken castor oil. The patient was practically an albino. At operation on the day of admission the whole appendix was found to be gangrenous and there was some free fluid present in the abdomen. During operation the appendix ruptured. After appendicectomy with drainage the patient's condition was moderately good. On November 10, 1926, he was fairly well and an enema was given with good result. On November 11, 1926, his condition appeared to be improving; on November 12 it was the same, though the abdomen was somewhat distended. At 4.15 p.m. the pulse rate dropped, the patient became unconscious and died in twenty minutes. No *post mortem* examination was performed.

CASE V.—Number 225, a female, aged fifteen years, is classified in the group "ruptured appendix with general peritonitis". She was admitted to hospital on February 23, 1928. According to the mother's account, the child, on January 25, had pains all over the abdomen; on February 8 she had a rigor and took castor oil. Vomiting followed, and two days later she had diarrhœa with mucus. At the same time she had frontal headache and pain and stiffness in the back of the neck. On February 11 a medical practitioner was called in. The child had a sore throat with white patches and was still vomiting. On February 14 the mother was advised to send the child to hospital, but she refused and kept feeding her on milk and milk foods. Two days before admission to hospital the child had a rash on the face and neck, diagnosed as a toxic erythema with absorption. On February 24 operation was performed, a gridiron incision being made. There was much oozing in the abdominal wall and pus was found on opening the peritoneum. A drainage tube was inserted and the wound was stitched. The patient died at 3 p.m. on February 25, 1928. The pus from the wound was found to be sterile when culture was attempted.

At *post mortem* examination a considerable quantity of dark blood was found in the abdominal cavity; much of it was clotted, and in the right iliac fossa a white clot was present. There was a considerable quantity of fluid blood in both kidney pouches and blood all over the small bowel and omentum. The appendix was thickened, kinked and embedded in an abscess cavity. The extraperitoneal tissue under the wound was blood-stained. There was a hole one centimetre in length in the peritoneum (made for the drain). No extraperitoneal clot was present. A loop of the bowel was very adherent and twisted, and the pelvis was shut off. At several places the small bowel was definitely obstructed.

According to the notes of the case, on February 24, 1928, at 11 p.m., the patient had been restless. Morphine was given. On February 25 she had had abdominal pain. She then felt better, the pulse was softer and its rate was 115. There was a sero-sanious discharge. At 1.20 p.m. the patient suddenly became worse and was in considerable pain. The pulse was imperceptible. "Hemostatic serum" and morphine, 0·0075 gramme (one-eighth of a grain), were given. The patient died at 3 p.m.

I was informed that this patient and her sister had both bled profusely after tonsillectomy. There is no doubt that the bleeding either caused or at least was a big factor in the lethal issue. It had been noticed that the abdominal wall bled very much, and meticulous care had been taken in tying the smallest vessels. The exact origin of the blood was indeterminable. Probably it came from several points.

CASE VI.—Number 249, a male, aged seventy years, is classified in the group "ruptured appendix with general peritonitis". He was admitted to hospital on August 4, 1930. He had been ill for five days. He had had iritis and glaucoma nine months previously. According to the history obtained from a relative, he had had indigestion and flatulence for five years. He suffered from vomiting every third month and had had ten days of epigastric pain. For the last five days there had been frequent vomiting, and micturition was frequent day and night. On examination the patient was found to be a very sick old man. Distension was present, and the urine, on examination, was found to contain a considerable amount of albumin. The patient died at 11.30 a.m. on August 5, 1930.

On *post mortem* examination half an inch of intact appendiceal tissue was found. The remainder was thick creamy pus. There was also free pus in the pelvis and the bowels were matted with fibrin.

The condition of this patient was not diagnosed until the *post mortem* examination was made. There was no operation.

CASE VII.—Number 254, a male, aged sixty-two years, is classified in the group "ruptured appendix with general peritonitis". He was admitted to hospital on November 11, 1930. He had been ill for two days. On the day of his admission to hospital appendicectomy was performed and drainage was instituted. The appendix was found to be gangrenous and ruptured. There was free pus, and the omentum and bowel were adherent; there was lymph over the intestines and œdema of the abdominal wall. Convalescent serum was given several times, and also gas gangrene antiserum. A 10% solution of glucose was given intravenously. On November 15, under local anæsthesia, jejunostomy was performed and a Paul's tube was inserted for drainage of the bowel. On November 23, 1930, the patient died.

CASE VIII.—Number 469, a painter, aged thirty-one years, is classified in the group "appendicitis with abscess". He was admitted to hospital on September 1, 1920, under the care of a physician. He had been ill for five weeks and before admission to hospital had been treated for lead colic. Five weeks prior to his admission to hospital he had been seized with severe abdominal pain, colicky and griping; he had suffered from mild attacks over a period of twelve months. There was no blue line on the gums. The patient was seen by me on September 5, 1920.

The patient's condition was diagnosed as being suppuration in the liver, possibly in a hydatid cyst. Laparotomy was performed on September 5, 1920, and the liver and the round ligament were found to be œdematous. Exploratory needling of the liver was performed and an operative diagnosis of pyelo-phlebitis was made. The patient died on September 20, 1920.

At *post mortem* examination the appendix was found to have sloughed off and to be floating about in the peritoneal cavity. Pyelo-phlebitis was also present. It was unfortunate for this patient that his occupation was that of a painter.

CASE IX.—Number 528, a male, aged sixty-four years, is classified in the group "appendicitis with abscess". He was admitted to hospital on January 5, 1922, having been ill for three days. He came from Helensburgh. He had had pain for two years and had been vomiting for three days. On January 5, 1922, appendicectomy was performed and drainage was instituted. Free pus was found. The appendix was gangrenous and broke off when touched; it was removed piecemeal. The patient's condition was not good and he died on January 10, 1922, having a short fit before death.

At *post mortem* examination, on January 12, 1922, anthracosis was found and congestion at the bases of both lungs. The peritoneum was normal and there was no fluid nor pus. At the base of the appendix stump there was some very friable slough, which was apparently shut off. The kidneys were both large, with minute hæmorrhages on the surface. A *post mortem* diagnosis of acute appendicitis with acute nephritis was made.

CASE X.—Number 546, a male, aged thirteen years, is classified in the group "appendicitis with abscess". He had been ill for two days when he was admitted to hospital on June 11, 1922. He had taken castor oil. Appendicectomy was performed on June 11 and drainage was instituted. There was a great deal of foul-smelling free fluid. The appendix was gangrenous and ruptured. Concretions were found lying free in the peritoneal cavity, and although the patient's condition was apparently good, he died on June 12 at twelve noon. There was no *post mortem* examination.

CASE XI.—Number 8,285, comes under the heading of "acute and chronic appendicitis". The patient, a male, aged sixty-four years, was admitted to hospital on March 5, 1929. There was no history. He was *non compos mentis*. At operation on March 5, 1929, appendicectomy was performed and drainage was instituted. The appendix was acutely inflamed, as were the cæcum and adjacent coils of ileum. The patient died on March 7, 1929. *Post mortem* examination revealed congestion and œdema of the lungs. In the abdomen about 30 cubic centimetres (one fluid ounce) of pus were found to be walled off. There were dense adhesions between the liver and diaphragm.

CASE XII.—This case did not appear in records under the heading of appendicitis. The patient was a female, aged sixty years, and was admitted to hospital on March 13, 1931, having been ill for six days. For twenty years she had suffered from a swelling of the abdomen, and for six days there had been pain round the umbilicus, with rigors. The bowels opened daily. There was a large ventral hernia reaching to within five centimetres (two inches) of the patellæ. On March 20, 1931, under local anæsthesia, a small incision was made and much pus was evacuated. Six or seven hours after operation the patient became cyanosed and died within fifty-one hours after operation.

Post mortem examination revealed a long appendix with a gangrenous tip in an abscess situated in a loculus of the left side. There was no general peritonitis. The patient had a large fatty heart with dilatation of the right side.

Reference.

(1) J. Colvin Storey: "Acute Abdominal Conditions", THE MEDICAL JOURNAL OF AUSTRALIA, April 2, 1932, page 465.

THE CAUSE OF DEATH IN HOSPITAL PRACTICE: A REVIEW OF 380 HOSPITAL AUTOPSIES.

By J. V. DUHIG, M.B. (Sydney),
Pathologist to the Brisbane Hospital.

THIS paper is an analysis of 380 *post mortem* examinations made of persons who died in the Brisbane Hospital. Some of the examinations were done at the order of the city coroner—a small number; most were on patients who had died in hospital after varying periods. The mass of material was so large that a statistical analysis on a large scale would be out of place in a paper designed for promoting a general discussion on the qualitative incidence of disease in the public hospital population, on the difficulties of diagnosis inherent in the material and in the conditions of hospital practice, and on the means to be taken to improve diagnosis and treatment. The paper is in no sense to be taken as a reflection on the skill of my colleagues, but is intended as a sympathetic though critical analysis made in a helpful spirit.

So as to come within a reasonable time limit and publication space, I thought it best to give first a bare statistical analysis and then a commentary in narrative form on special points. These are necessarily few, but I might suggest a plan I invariably adopt, that of reading up separately each condition as I see it in the *post mortem* room. The lists I give later of all these as I found them gives an extremely wide range of choice.

I am in the habit of saying, and there is no harm in repeating, that I never do a *post mortem* examination without learning something of permanent value from it; to be animistic, I might suggest that Nature has a most disconcerting way of presenting in almost perversely multitudinous combinations

[1] Read at a meeting of the Queensland Branch of the British Medical Association on April 5, 1935.

the unimaginably vast number of atoms at her disposal. The work is full of surprises to start with, but is so full of recurring microcatastrophes that she becomes tediously repetitive. But that is how humans learn, and it is this repetition of lesions in apparently new, but, I suppose, really very old, combinations of organic factors that lays the basis for the classical syndromes. I think it needs a very long and thorough experience in *post mortem* pathology to enable the worker to generalize masses of data into orderly diagnostic units; a suspicion of coherence of certain signs can be worked up into certainty only over series of hundreds. The solitary appearance of a thrombosis in a certain situation is almost devoid of significance unless *ante mortem* semeiology of the condition can be observed and the usual expectation of result confirmed or denied over a very large series. It was only in this way that appendicitis, for example, became common in the surgeon's mind, though it was unimaginably common in the bodies of mankind. But in spite of all our looking, we can never make vascular tumours anything but a great rarity above the *tentorium cerebelli*. So that only an enormous experience can enable a man to say what is common and what is not common in the way of disease.

I believe, and used to act on the belief, that it was a duty of medical men to report cases of interest and of rarity, either as a whole or as to some aspect. I have not ceased to believe that, only I have not the time to act on the belief. The reason for it is that the constant reporting of the repetitiveness of Nature will generalize in a big way what seem to the mind of the overworked general practitioner, looking for short cuts to diagnostic excellence, merely incoherent shreds in the rare tissue of fantastic syndromes, too fancy for an ordinary man to diagnose. I think that even now many practitioners feel it an outrage on the private decencies to have to admit to themselves and confess to their patients that they are suffering from the Pel-Ebstein syndrome or Ayerza's disease. That is not to say that these two diseases do commonly exist, though they never did before; I mean substantially in the mind of the doctor. It amounts to the idea that doctors should have the fullest knowledge of whatever news is going in the world of disease.

Now this analysis of 380 autopsies is my contribution for the time being. Some time ago I told you of the manner of death of those who die suddenly or violently, and it was a picture not greatly different from that found in comparable sections of the population elsewhere. What I am to give you tonight is about a different kind of population altogether, and the two sections bear no sort of resemblance or relation to each other, any more than the two reflect the fatal conditions in the general population. It is very difficult to say what significance is to be found in the figures and details I am going to give you, because the autopsies are not a random sample of the hospital population, nor are they strictly selected on any plan. They are conditioned by a number of things.

There is first of all the admirable curiosity of some resident medical officers at the Brisbane Hospital, from which the material is exclusively drawn, curiosity that is about death in general, and about, sometimes, possibly rare diseases in particular. Sometimes again the diagnosis before death was impossible or so difficult that no attempt but a very half-hearted one was made. Sometimes the coroner required accurate information, and sometimes I did an autopsy for the best of all possible reasons, because I let it be known that all patients should be examined after death, and the examination was one of this routine type, all the easier to get occasionally when the deceased person has had no relatives or friends to object. I think the very best use to which a dead body can be subjected is to be examined by a pathologist, but the only use made of some, at the express wish of relatives or friends, is to bury them. As an unrepentant and inveterate breaker of tabus I have tried to increase the amount of *post mortem* work at the hospitals that I have been connected with, and I have succeeded at the Brisbane Hospital because of the admirable zeal of the young men who come to us as resident medical officers, particularly those who have acquired a very persuasive technique with relatives while they were first resident medical officers in large clinical schools in the south. There is no royal road to getting permission for *post mortem* examinations, but only the plebeian one of asking. As an amateur anthropologist interested in the local and contemporary folk-ways, I often wonder why people object to autopsies on their relatives, and what is their idea of death, and whether they have any idea at all about how the maternal science of pathology was developed, that brought forth that of ætiology, which is so beneficent and which becomes more so only as its mother science is well served. In the extremely superstitious and poorly educated big children that form the main bulk of our populations, persistence of the belief in spirits of the departed as a very widespread mental phenomenon is only to be expected. Neither the theory of fear adopted by Tylor and partially by Frazer nor that of infantilism favoured by Freud completely explains that curious mixture of repugnance and respect and the quaint ceremonies connected with the dead and their disposal. The unwillingness to grant permission for autopsies is a very real obstacle to pathological progress. It may be superfluous and ostentatious to assure you that my interest is merely the promotion of knowledge, and if it is useful to profess reverence for the dead, I am ready so to profess and think that that is, for the present, the better attitude. But I think the reverence that denies other humans the benefit of sound knowledge only to be acquired by an examination of the dead is altogether excessive; it takes the pernicious colour of sentimentality, the inveterate enemy of truth and sound sentiment.

When I joined the staff of the Brisbane Hospital, *post mortem* pathology was little practised, because the work was left to the resident medical officers,

and they showed the usual lack of enthusiasm for work about which one knows little and for which one has not the training, so that it becomes a tedious corvee. In one year three *post mortem* examinations were made. I have not been able to discover the reason for this: why they were done at all. The institution has been so chronically understaffed that there was never any time to do the work properly, if at all.

I have kept records of the work since the staff meetings were resumed in a businesslike way, and the percentage of autopsies on deaths was 20, 35, 25 for the first three two-monthly periods, 32 for the next four months, and 31 for the next twelve months after that and down to the end of the period covered by this paper. The percentage of the 380 autopsies analysed on the deaths for the period (1,287) is just barely short of 30%. That is a great improvement on former figures, which were microscopic occasionally and always small, but it is only a third of what it should be. I cannot insist too much on the value of autopsy work, and, whether the clinician sees the operation or not, he gets eventually a wonderful return.

Before going on to analyse the returns, I am sure I am filling a long-felt want existing in the minds of all of you when I tell you of the measure of success of the clinician's diagnosis before death. I have worked this out on the simple plan of comparing the clinical diagnosis with the autopsy findings under the rubrics "Agreement", "Partial Agreement", "Disagreement", "No Diagnosis". It should be stated at once that the *post mortem* pathologist, contrary to the usual idea and his own early assumptions, is not infallible. Not only is his interpretation liable to be faulty, but he cannot find what is not there to be found. I must confess that many children die from causes which still remain to me a complete mystery after the autopsy, and that after convulsions, many nervous diseases and hyperthermias no good reason is apparent why the patients should have died. Many excessively conscientious pathologists place the number of their unsuccessful autopsies very high; I am not so modest, as you know. Some partial correlation of the living and dead pathology is nearly always possible, though a complete explanation of events is sometimes missing. Still *post mortem* diagnosis is vastly more accurate than the clinician is entitled to expect for himself. Many astronomers would like to dissect the moon, but must, for the present, remain content to admire it from afar.

Out of 380 autopsies analysed, the facts correlated with the *ante mortem* diagnosis showed: "Agreement", 158 (41%); "Partial Agreement", 76 (20%); "Disagreement", 144 (roughly 38%); "No Diagnosis *ante mortem*", 2 (under 1%).

It must again be remembered that the autopsies were done mainly on doubtful or very difficult cases and the accuracy of *ante mortem* diagnosis in general is considerably better than 41%.

Contrary to what would, I suppose, be the normal expectation, diagnosis of intracranial conditions was uniformly good, mainly because it turned out to be in fact very easy. In the long list, setting out all the conditions found, it will be seen that on the whole those connected with the skull and its contents were fairly straightforward. Even then the successful diagnosis of brain tumour was surprisingly high. It was practically never missed, though so far our clinicians have not yet got to the stage of identifying the kind as well as the site of the tumours found. This is not so difficult to do, as apparently Cushing and Bailey believe the tumours run true to type in the various situations. I have noted the fact that genuine vascular tumours above the tentorium are so rare up to the present at least that a cerebral tumour can be said not to be of that kind. There is one condition that is extremely common, however, and is rarely diagnosed, namely, cerebral œdema, which is usually lumped in with the syndrome conveniently but inaccurately called uræmia. This fact was responsible for much of the partial agreement in *ante* and *post mortem* diagnosis. I shall have much more to say about that later on.

Diagnosis of thoracic conditions was intermediate in accuracy between cranial and abdominal. The table shows the marked preponderance of the lungs as the seat of the fatal lesion and the enormous variety of conditions found, which, I can only conclude, give inevitably equivocal physical signs. The interpretation of these is evidently a matter of opinion and lacks the precision of the positive findings of the laboratory worker, which it is the fashion to pretend to despise nowadays. Why it should be more estimable to be a little uncertain on physical signs alone whether a patient is suffering from tuberculosis or cancer of the lungs than to be sure on the ground of a positively accurate bacteriological finding, I find it difficult to understand.

Diagnosis of abdominal conditions is apparently very difficult and is best only when supported by positive physical, chemical or biological data, such as the evidence of a test meal in the differential diagnosis of cancer of the stomach or pancreas, about which I shall have more to say in the key of my opening remarks, and the hyperglycæmia of *diabetes mellitus*.

The mistakes in diagnosis were most often due to inherent difficulty insuperable in the present state of clinical medicine, as when a patient is admitted unconscious, no real history is available and there are no extrinsic data to help. This sort of thing is not uncommon in accident cases, when the cause of unconsciousness may be concussion or hæmorrhage, a ruptured viscus or just profound shock. Quite commonly, as I shall show later, pyonephrosis, bilateral and literally of enormous extent, exists quite unsuspected right up to the time of death. Theoretically this should be easily diagnosable at this stage, but it is precisely at this stage that the patient is too ill to be tampered with. Later on I shall suggest some more detailed attention to this condition in the early stages. Sometimes the real

lesion is one which it would have been ostentatious to suggest, even if it had been possible to arrive anywhere near it with our present means of clinical investigation. Such a lesion was a primary malignant plasmacytoma of the spine with secondary deposits in the pancreas and myocardium, amongst other places. The clinical diagnosis was set down as "infected buttock for investigation". Sometimes again a common enough lesion is not sufficiently in the mental view of the clinician and is at the same time not at all a distinctive syndrome, such as carcinoma of the pancreas, about which I shall have more to say later. A common diagnosis on rather old-fashioned traditional lines is that of asthma, sometimes promoted to the doubtful dignity of cardiac asthma, when something else besides a true allergy is meant or present in the patient. I suppose every generation is cumbered with traditional inaccuracies like this one, and since text books are usually many years out of date in some details before they are actually printed and distributed, it is inevitable that a lot of traditional error should survive longer than it should. Some diagnoses are so fantastically wrong that there is a sound reason for them. Some such are: (i) Intestinal obstruction diagnosed in a case of coronary occlusion, evidently the second attack of the sort since there was adherent pericardium over the site of an old cardiac infarction. (ii) "Localized Paget's disease" diagnosed because of a bony lesion in the humerus of the left side, which turned out to be a secondary deposit of carcinoma from a primary growth in the pancreas. And (iii) probably one of the most fantastic diagnoses ever made, "carcinoma of the colon" in a case of coronary thrombosis. This diagnosis was made twice in this series in exactly similar circumstances. It is well known, of course, that a cardiac catastrophe of this kind may, to the casual clinical observer, simulate ruptured gastric ulcer, cholecystitis, cholelithiasis or acute dyspepsia of any kind, but I find it difficult to understand why a cancer should be incriminated, except that an intestinal obstruction, for example, must have some cause, and that, for a conjectural one, cancer is as good as any and may turn out a brilliant inspiration. I have not much faith in inspiration myself, since I have seen immeasurably better results come from care and average skill.

I have not the slightest intention of ridiculing my colleagues' efforts at diagnosis; I merely wish to point out the great difficulties of an art which is very strictly limited in its scope by the necessity for safety and personal consideration of the patient and the impossibility of inflicting manipulative harm beyond what the patient already suffers.

Another common source of error is the occurrence of the first few sporadic cases of a disease which is to be later epidemic or at least very widespread, such as poliomyelitis. One case of this disease I missed myself at the beginning of the last epidemic. As the presence of the disease is made known by the public health authorities, the diagnosis becomes much easier than the average case of any other disease.

Still another is that the leading symptom directs attention to an organ which is subject to secondary damage by reason of impaired function in another. The pain of a liver tensely enlarged by the chronic passive venous congestion consequent on a failing heart not seldom prompts a diagnosis of cholecystitis, and in one such case the patient was operated on and, of course, died. The pallor of myxœdema another time led to a suspicion of pernicious anæmia, and the patient's condition was so advanced that her survival in any case would have been doubtful. A long search failed to reveal anything but the faintest shreds of vestigial thyreoid gland. And to complete a partial tale of failure, except in the brain, the relatively common lesion of vascular damage and thrombosis and embolism is hardly ever thought of. I would not say that this kind of lesion is commoner than it was, but I find it not uncommonly, and surprisingly little reference to it is made in a systematic way in the books. I think the vascular basis of disease is much wider and more important than is thought.

The accuracy of diagnosis is analysed in Table I according to the site in the body of the fatal lesion—head and neck, thorax and abdomen. A large number of diseases could not be strictly fitted into this scheme, such as septicæmia or diabetes, and these are shown under the head of general. Complete agreement between the *ante* and *post mortem* diagnoses is shown as A, disagreement as D, and partial agreement as P. Under the totals of each heading the percentage figure is given on the complete total of each system.

TABLE I.

Head and Neck.			Thorax.			Abdomen.			General.		
A.	D.	P.	A.	D.	P.	A.	D.	P.	A.	D.	P.
44	21	11	39	42	24	39	64	26	34	14	12
58%	28%	14%	37%	40%	23%	30%	49%	21%	56%	23%	21%

It will thus be seen that success in diagnosis is directly proportional as we proceed from above down, and that abdominal diagnosis seems to be very difficult. The success in the diagnosis of general conditions was due to the inherent simplicity of diagnosing septicæmia by blood culture, and diabetes by blood sugar estimations. Unaided clinical diagnosis varies in success with the complexity of the organic system involved. I should like to insist again on the fact that this apparently poor result does not apply to clinical diagnosis in general, about which I have no data and upon which I am completely unqualified to speak. The cases in the series under review were examined *post mortem* precisely because the clinician acknowledged uncertainty or defeat for a very good reason, such as the inadvis-

ability of making a more than cursory examination of the patient in his own interest, and other equally cogent reasons.

It cannot be claimed that the autopsies analysed here represent a cross-section of all the deaths or all the clinical conditions occurring in the hospital, but in so large a series as 380, representing nearly one-third of all the deaths, they must be in some sense representative of the kind of difficulty met with and of the special nature of the difficulty. It is instructive, for example, to compare the fact that half the diagnosis in abdominal conditions was wrong and another fifth partly wrong with the conditions actually found.

So as to give some idea of the conditions met with, I give the complete list of those I found, grouped under heads corresponding to the principal organ involved or type of disease or manner of death. Since some of the diseases involved more than one organ or mode of death, such as death by accident from fracture of the skull, which I list under "Accident" and possibly also under "Cerebral Thrombosis", the total figures in the list will be found to outnumber 380, or the number of bodies examined.

The lesions found under the heads stated were, then, as follows:

Lungs.

Tuberculosis, 16.
Silico-tuberculosis, 2.
Lobar pneumonia, 16.
Bronchopneumonia, 11.
Abscess, 4.
Cancer (primary), 2.
Bronchiectasis, 2.
Empyema, 3.
Pertussis pneumonia, 2 (ages 14 months, 22 months).
Embolism, thrombosis, infarction, 7.
Pulmonary œdema, 17.

Heart.

Coronary thrombosis, occlusion, infarction, 13.
Aortic incompetence, 6.
Rheumatic endocarditis, 3.
Malignant endocarditis, 1.
Aortic aneurysm, 4 (2 ruptured).
Syphilitic aortitis, 2.
Mitral incompetence, 2.
Myocardial degeneration, 5.
Rheumatic carditis, 2.
Rupture of coronary aneurysm, 1.
Chronic adherent pericarditis, 3.
Acute pericarditis, 3.
Suppurative pericarditis, 2 (both pneumococcal).
Auricular fibrillation, 2.
Acute heart block, 1.
Alcoholic fatty degeneration, 1.

Brain and Membranes.

Cerebral hæmorrhage, 22 (1 basilar, 2 cerebellar, 1 pontine, 1 anterior cerebral, the rest lenticulo-striate).
Cerebral thrombosis (vascular), 6 (2 from fracture of the skull).
Cerebral thrombosis (septic), 3.
Cerebral tumour, 6 (1 cerebellar, 1 metastatic melanoma).
Cerebral abscess, 3.
Tuberculosis, 4 (1 tuberculoma).
Acute meningitis, 8.
Leptomeningitis, 1.
Concussion, 3.
Epilepsy, 1.
Subarachnoid hæmorrhage, 2.
Measles encephalitis, 2.
General paralysis, 1.
Bulbar palsy, 1.
Acute encephalitis, 1.
Cerebral œdema, 6.
Cerebral œdema, with chronic internal hydrocephalus, 2.

Digestive Tract.

Cancer, 13 (5 stomach, 3 œsophagus, 5 colon—4 in the sigmoid).
Rupture of ulcer of stomach or duodenum, 4.
Acute dilatation of stomach, 1.
Thrombosis of gastric veins with gangrene, 3.
Ileus, 3.
Volvulus, 2.
Acute hæmorrhagic enterocolitis, 3.
Tuberculosis, 1.
Abdominal lymphosarcoma, 1.

Liver.

Cirrhosis, 9 (1 syphilitic).
Cancerous cirrhosis, 1.
Abscess, 2.
Hydatid disease, 1.
Acute necrosis, 1.
Alcoholic degeneration, 1.
Cholelithiasis, 1.
Empyema of the gall-bladder, 1.

Kidney.

Nephrosclerosis, 22.
Pyonephrosis, 16 (2 bilateral, 2 calculous).
Cancer, 1.
Hydronephrosis, 1.
Chronic nephritis, 2.
Multiple embolic abscess, 1.
Chronic nephritis with pyelitis, 1.
Tuberculosis, 1.
Bilateral polycystic kidney, 1.

Pancreas.

Cancer, 17.
Cirrhosis, 1.
Acute necrosis, 1.
Chronic pancreatitis with lithiasis, 1.

Blood.

Pernicious anæmia, 4.
Leuchæmia, 5 (myeloid 3, lymphatic 1, myeloblastic 1).
Cholæmia, 2.
Purpura fulminans, 1.
Neutropenia (agranulocytic angina), 1.

Bones.

Sarcoma, 1.
Plasmacytoma, 1.
Suppurative polyarthritis, 1.
Suppurative monarthritis, 1.
Chronic osteomyelitis with amyloid kidney, 1.
Osteomyelitis producing pulmonary embolism, 1.
Tuberculous joint, 1.

Uterus et cetera.

Abortion (sepsis *et cetera*), 7.
Rupture, 2 (instrumental 1, traumatic rupture of gravid uterus 1).
Eclampsia, 1 (associated with pituitary adenoma).
Cancer, 2 (uterus 1, ovaries 1).
Pyosalpinx, 1.
Myoma producing anæmia, 1.

Vessels.

Thrombosis of branches of arch of aorta, 1.
Thrombosis of common iliac artery, 1.
Thrombosis iliac vein, 1.
Thrombosis of gastric veins, 3.
Thrombosis of mesenteric veins, 3.
Œsophageal varix, 1.

Bladder et cetera.

Rupture of the urethra, 1.
Cancer of the prostate, 1.

Peritoneum.

Acute and suppurative peritonitis, 13.

Mental.

Acute mania, 1.

Infection.

Septicæmia, 7 (2 abortions, 1 puerperal).
Gas gangrene following abortions, 2.
Carbuncle, 4.
Cellulitis, 2 (face, rapidly fatal, 1).
Lung abscess following carbuncle, 1.
Diabetic gangrene, 1.
Pertussis, 2.
Arteriosclerotic gangrene, 1.
Tetanus, 1.
Typhoid fever, 1.
Peritoneal gangrene from thrombosis, 1.
Acute *otitis media*, 1.

Anæsthetic and Post-Operative.

Appendicectomy, 1.
Cholecystectomy, 1 (pulmonary thrombosis).
Peritonitis, 1.
Breast amputation, 1 (N_2O-O_2).
Fracture of the leg, 1 (shock).

Suicide.

Hanging, 1.
Coal gas, 1.
Cyanide, 1.
Strychnine, 1.

Accident.

Fracture of the skull, 22.
Fracture of the spine, 3.
Rupture of viscera, 4.
Rupture of uterus (gravid), 1.
Rupture of vessels of neck, 1 (blown up by dynamite).
Rupture of intercostal by fractured rib, 1.
Others, 8.

Constitutional.

Diabetes, 8 (2 with gangrene).
Acute alcoholism, 1.
Chronic alcoholism, 2.
Filariasis, 1.

Cancer.

Pancreas, 17.
Stomach, 5.
Colon, 5 (4 sigmoid, 1 ascending).
Œsophagus, 3.
Larynx, 2.
Lungs, 2.
Lymphosarcoma, 2 (tonsil, abdomen).
Kidney, 1.
Uterus, 1.
Ovaries, bilateral, 1.
Prostate, 1.
Melanoma, 1.
Malignant myoma, 1.
Multiple plasmacytoma, 1 (primary in spine).
Lympho-epithelioma of nasopharynx, 1.

Miscellaneous.

Myxœdema, 2.
Goitre with bronchopneumonia following obstruction, 1.
Intracranial suppuration, 2.

With a few unimportant omissions, that is the list of the lesions found in the 380 autopsies studied.

There are some special features of the work and the results obtained that I should like to mention. The order in which I take them is the convenient one suggested by the order of the list of lesions, simply that and no other.

After the external examination and opening the body I inspect the thorax first. I have commented in another paper read before this Branch on the importance of the degree of calcification of the costal cartilages. I still record this fact. There are four special points I wish to mention in connexion with the lungs. There are no doubt others, but as this is not a systematic treatise on *post mortem* work, but a record of my own impressions and fancies, I omit others which do not appeal to me so much for putting before an audience in a special way.

I should say, however, that I was surprised to find that of the sixteen patients who died of tuberculosis of the lungs, all but one were over fifty-five years of age. Nearly all these patients died with their condition undiagnosed, on account, I suppose, of their age. This active tuberculosis at so advanced an age raises the question of self-reinfection from a lesion acquired in childhood or a new infection altogether. I may say that all the lesions were the usual chronic phthisis with lymphatic spread.

The four special points are: (i) pleural adhesions, (ii) lung abscess, (iii) pulmonary thrombosis, and (iv) pulmonary œdema. As I told you on a former occasion, healed or quiescent tuberculosis of the lungs is so common that I just record it and think of it no more.

I have not seen it elsewhere noted that pleural adhesions are of two kinds, congestive and inflammatory. The former are fine, friable and easily detachable, and for obvious reasons are fresh and recent, because pulmonary congestion does not last long before recovery or death. The latter are firm and organized and detachable with difficulty. The congestive adhesions are associated with œdema and follow an exudate poor in fibrin; the inflammatory adhesion is richer in fibrin and so organizes more quickly and more firmly. Whether this observation is of any use in clinical medicine, I leave it to yourselves to decide.

The kind of lung abscess I hate to see, because it should be so unnecessary, is that following teeth extractions under general anæsthesia. If they do not end in death, they produce bronchiectasis. This particular complication is shown as occurring in a fatal form in only two of the series, but it is in fact relatively common, at least to me. I regard as bronchiectatic all those small saccular, fusiform or even tubular dilatations of the bronchi, especially in the lower lobes, so commonly found, in which the calibre of the tube is clearly wider than normal or than the corresponding tube on the other side, and, necessarily as an accompaniment, with peribronchial fibrosis and loss of elasticity. I think this is very common, and I mention it so that its causation may be discussed.

Of all the catastrophes, commoner than thought, the worst, or at least the most disturbing, is embolism or thrombosis of the lungs, whether it is fatal or not. Coronary block is not so upsetting, because the public seems to expect heart failure sooner or later, and it occurs quite independently of any other lesion; but lung embolus usually occurs while the patient is under treatment for something else, not seldom after an operation from which he is a long way on the road to recovery. One such case in this series occurred in a woman after a clean and altogether successful cholecystectomy. I took out of the lungs, particularly the left, a firm, hard, dry, thrombotic cast of the pulmonary circulation, spreading right back to the left auricle from a tiny saddle embolus right out near the visceral pleura. These emboli are sometimes hard to find, but if the technique of Belt, of Toronto, is followed, not many will be missed. This is to open the lung, outside the body, of course, along the vertebral border, along a vertical plane close to the root of the lung, when the pulmonary circulation will be laid open in such a way that it can be very easily and carefully followed down without disturbing any clot, however small, in the circulation. It is worse than useless to try to track a thrombus up from the surface of an infarct. I find that pulmonary thrombosis is responsible for only about 2% of deaths in this series, but I feel that up to about eighteen months ago I was not so careful in searching for the lesion as I am now. That is an example of the sort of intellectual process I spoke of in the beginning, that as one begins to notice repetitive phenomena casually, they become very common to a mind in a better state of awareness. I should say, then, that this accident would be more frequent than I represent it, by how much, though, I dare not say. It is in any case a most serious problem to those treating and nursing patients with sluggish circulation and any kind of varicosity. Belt says he finds crural, hæmorrhoidal or pelvic varix invariably in his interesting series of pulmonary thrombosis, but I have been not so fortunate. If he is right, at least one leading principle of prophylaxis is established. I allude to this matter with considerable feeling, because death in this way is so tragic and discouraging and occasionally disastrous to the prestige of good surgeons; even more so, perhaps, is non-fatal embolism during convalescence. Perhaps I owe an apology to my Brisbane Hospital colleagues for being, to them, unduly tedious about this matter.

Pulmonary œdema is also a disheartening complication, especially if it be very acute. In this series I have mentioned it only as it occurred as a really significant factor in the death. If it is looked for, it will be found very frequently, though it does not seem to be of much importance to the clinician. I may be wrong in this, but, looking over the physical signs of numberless cases and comparing them with my findings, I am surprised at the insignificance of the condition to the clinician. In kidney disease, failing heart and any other con-

dition which may produce it, it must be extremely difficult to deal with, since it must cause very serious embarrassment to the patient on account of the vicious circle set up involving the circulation and deficient oxygenation. In this series I find that it occurred really acutely only once in a patient suffering from hyperpiesis.

It is significant that there are only four cases of endocarditis in the series. I am quite sure, from my knowledge of the bacteriological side of my department's work, that infective endocarditis is relatively common. I touch on the topic here to say, however, that I have seen much less endocarditis confined solely to the mitral valve than to the aortic, alone and in combination with the mitral.

Myocardial degeneration occurred five times, two of these being associated with coronary disease. The old authors constantly referred to myocardial disease and very often attributed the lowered function to fatty degeneration, confusing this condition with the fatty infiltration that occurs in obesity and alcoholism. There is no doubt that a certain amount of fatty change must occur in the cells of the myocardium in old age or at any age if oxygenation is defective as a result of interference with the blood supply. I do not think, however, that the amount of fatty degeneration is ever sufficient to account for death on that ground alone. Ischæmia in its widest sense, with infarction or heart failure, will occur first and cause death before the fatty change has a chance to have any serious influence on the outcome. I have not seen fatty degeneration of the myocardium as a serious factor in a death, except once, and that was as part of a generalized fatty change in a hopelessly alcoholic subject. But that the myocardium degenerates in function is quite undoubted, owing, I believe, to an underlying vascular defect. In the last paper which I presented to the Branch I showed a stereoradiograph of an old heart studded with multiple miliary calcareous nodules, which I take to be areas of old fibrosis following minute infarcts at the site of impaction of tiny emboli or capillary thrombosis in the heart muscle. Sometimes I see large areas of fibrous replacement at the site of large infarcts, and it is very common to see a fibrosis at the tips and extending down the papillary muscles, due undoubtedly to the fact that the arteries of the papillary muscles are end arteries or at least have a very poor anastomosis, and that so small local infarctions are not readily overcome. I do not think this papillary fibrosis is of much significance, as I notice that the amount of incompetence of the mitral valve due to contraction of the muscles is quite small and an otherwise sound myocardium will hypertrophy to the required extent. But the implications of the observation are more interesting. These tiny focal emboli must surely occur very frequently right through life, from the fourth decade onwards, since atheroma is almost universally present in humans living at least in conditions similar to those here. I suppose those rather vague pains in the chest over the heart must be due to something of the sort.

It will be noticed that one diagnosis under the heart heading is "Acute Heart Block". This was made on the history more than on the findings; it is part of the logical outcome of my interpretation of the morbid anatomy of the circulation of the heart. I believe that that part of the conduction system known as the bundle of His, being supplied almost exclusively on both its right and left branches by the left coronary, may suffer acute cut-out by vascular block. What we normally see is only partial block, depending on survival of at least some small part of the blood supply.

Cerebral conditions seem to be on the whole very straightforward, and comment is called for in only a few of them.

Cerebral concussion is apt to be puzzling, and, even if it is diagnosed, it is difficult to know what to do, since the amount of bleeding accompanying it can only be a matter of conjecture or of waiting until intracranial tension is so raised as to leave no doubt of the amount and situation. But even then localization is apt to defy all diagnostic systems. I am assuming it as a commonplace that concussion as a cause of much intracranial hæmorrhage and death is recognized. It is sometimes said that brain destruction always accompanies concussion; that has not always been my experience. Deaths from this cause are most often associated with shock and hæmorrhage.

I next merely direct attention to two deaths from measles encephalitis. The cerebral involvement in these two cases both before and after death was very evident. The measles epidemic last season was particularly severe in the Infectious Diseases Hospital at Wattlebrae.

In the early part of this paper I made reference to the term uræmia. To judge by the amount of urea the organism can tolerate over the normal, it cannot be a very poisonous substance. Assuming 30 milligrammes *per centum* to be a normal figure, 15 times that amount, 450 milligrammes, still does not produce death. Of course, animal analogies do not always hold with humans, but it is interesting that Professor Dakin, of Sydney, has shown that marine animals normally have, in comparison with humans, enormous blood urea contents. It is evidently not poisonous to animal protoplasm. On the other hand, most old men who choose to die that way rarely have a blood urea content beyond 100 milligrammes, but, being, as it were, loose-tissued, they easily get œdematous, especially in the brain. These old brains in men with arteriosclerosis, poor excretion and failing hearts are always wet and diffluent. It is the œdema which is responsible for all the intracranial symptoms, and whether that is of any assistance to the clinician, I do not know. At least he can be more correct and use the term on the death certificate instead of uræmia in future. The terminology is really bound up with the whole question of what is called nephritis here. That term so dominates the clinical outlook that the word

uræmia naturally is used. It should be dropped in any case, and particularly because the kidney symptoms and changes are in most cases really vascular in origin. We shall return to that later.

It was in the digestive tract that I first found the most interesting lesion that I think I ever found; and, in that curiously rapid recurrence of the experience, I noted a very common phenomenon: that strange things come in groups and are seen but rarely afterwards. I saw, for example, two cases of bilateral pyonephrosis in infants, probably congenital, in a week. I shall be surprised to see another case in the next ten years.

The pathological curiosity I saw in the stomach and saw again within a very short time was gangrene of the stomach following thrombosis of the gastric veins; in the first case the right gastro-epiploic and pancreatico-duodenal veins were involved. They were hard and tightly thrombosed, like cords, and the stomach tissue in the area was gangrenous. This is related as a curiosity, as I said; it is not suggested as a common diagnosis; such a diagnosis would be rather ostentatious.

Under diseases of the liver we note only one instance of hydatid disease, which about represents its frequency here.

Under the heading of the kidney we note twenty-two cases of nephrosclerosis and only two of genuine chronic nephritis. This classification is founded not only on the macroscopic examination of the organ, but on sections in almost every case. The deaths occur at all ages, and no distinction is made between the strictly arteriosclerotic kidney and the senile type following *endarteritis obliterans* and frequently cortical cyst formation, due, of course, to that vascular abnormality. The disease is primarily vascular in origin and all the cases are grouped together on the ground of this common origin. It is opportune just here to say that in the causes of death in the long list I submit, I do not even mention arteriosclerosis and atheroma, the two great enemies of man. The list is one of pathological states found in organs, and these states are often the result of these two vascular states. Atheroma is universally present at and after middle age, but does no great harm until the sixth decade as a rule, when it produces coronary block of some kind.

Arteriosclerosis, on the other hand, occurs at all ages from the second decade onward, though, of course, it is much commoner at and after middle age; and it is this disease of the vessels which is responsible for the high incidence of kidney insufficiency in this State of Queensland in the early years of life as compared with the later incidence in other States of Australia. Every case of cerebral œdema was associated with it in this series and a considerable amount of pulmonary œdema. The solution of the pathological problem of atheroma with its dread sequel of coronary disease, and of arteriosclerosis with its kidney failure and cerebral œdema and cerebral hæmorrhage, will do very much to advance the age at death, if that is thought to be desirable. I should think it desirable for the adolescent and adult and early middle-aged people who now die without hope before their time.

A disease which seems to be extremely difficult to diagnose is pyonephrosis, of which sixteen cases were found in the series and none of which was diagnosed, often for the reason that the patient was so ill on admission to hospital that examination could not be at all adequate. Something should be done about this group. Very often the patients are not much past middle age, round about fifty as a rule, and so much damage has been done to the kidneys that they are quite literally beyond repair. The disease must be very slow and insidious, both in onset and course, and the patients seem to be able to accommodate themselves to whatever discomfort there is. I show tonight a specimen of the condition which is the best example that I have seen of what a human being can stand. The kidneys are just shells of tissue, the ureters are thicker than the biggest abdominal aorta, and the bladder was enormously dilated. What must be the early symptoms and signs of these cases and how can the patient's attention be drawn to them so that people will take advice about them? Is there a congenital defect that might lead to a prediction of later trouble, such as hydronephrosis, to which the patient becomes accustomed from his very earliest years? Apart from this problem of prevention, pyonephrosis is always a good diagnosis to try in a case of abdominal disease of obscure origin in a middle-aged patient. I should also say that practically all the cases were in men.

Cancer of the pancreas seems also to be very difficult to diagnose. It occurred seventeen times in this series and was diagnosed only once. I have dealt with the disease in my presidential address to the Section of Pathology at the Australasian Medical Congress at Hobart in 1934, and an exhaustive analysis of all the data relating to nearly forty cases is there given. That analysis shows that there is no sign or symptom which can surely be referred to the disease alone. All I can suggest is that a cachectic state, probably arising from an abdominal neoplasm, with symptoms of upper abdominal involvement without enlargement of the liver is likely to be associated with a pancreatic cancer. A rapidly increasing jaundice will confirm the diagnosis, but too late to allow anything to be done. In any case, what can be done? The only fact now worth noting is that *post mortem* examination has served to make the records in respect of this disease accurate. Formerly this disease was probably diagnosed as cancer of the stomach. And seventeen cases since July, 1933, is a formidable total.

Still under the heading of the pancreas, I note one case of acute necrosis. I use this term in preference to the old one of acute hæmorrhagic pancreatitis, as being more accurately expressive of the actual state of affairs.

There is nothing of great importance to note under the heads of blood and bones, except the rare conditions of neutropenia and plasmacytoma, the latter of which I shall deal with in a longer communication elsewhere.

One lesion of most especial interest is the case of eclampsia. You will remember that Cushing enunciated the theory that high arterial tension and such allied states as eclampsia were associated, causally possibly, with basophile cell invasion of the *pars nervosa* of the pituitary, not necessarily neoplastic. It is most curious that the first and only case of eclampsia I came across after reading one of his papers on the subject did have a basophile adenoma of the pituitary in the posterior lobe. I naturally, in so large a *post mortem* practice, have had ample opportunity to test Cushing's theory and, so far as hyperpiesis is concerned, it is not true, nor, so far as I have gone, is it true in regard to the pituitary glands of those women dying of the nephritis of pregnancy. These cases are very few no doubt, but they have not yielded a single confirmatory instance. These deaths are not recorded here, since they occurred at the Lady Bowen Hospital and could not be included.

The deaths recorded under the head of infection do not, of course, represent anything like the mortality due to this cause. The only items of note are the case of cellulitis of the face, in which the patient came to attention too late to have his angular vein tied, and the two cases of gas gangrene following abortion. There was only one death from typhoid fever to be examined, and that, I think, about represents the severity of the disease here. I suppose the clinical staff were so shocked at losing a patient with typhoid fever that they decided to look and to be sure of the diagnosis.

The anæsthetic death was, curiously enough, under the kind of anæsthetic which was considered to be so eminently safe, nitrous oxide and oxygen. Two immediately post-operative deaths were due to pulmonary embolism. One death was due, I think, to hasty operative interference with a compound fracture. This is the only death in the whole 380, which, I think, should not have occurred.

In the accident series the most curious and unfortunate death was due to a ruptured intercostal artery divided by a fractured rib. The hæmorrhage was not discovered.

The last lesion I wish to discuss is that of filariasis associated with a fever. It is the first and only case I have seen, and the amount of fluid in the retroperitoneal tissues was most remarkable. Being thoroughly unfamiliar with the manifestations of the disease since it started to decline here, I had to rely on book description for guidance, and the lesion seemed to tally with a rupture of the great lymph trunk of the abdomen. Just why these people die escapes me.

That is the retrospect of these 380 deaths.

Examining the bodies and going through the records has been a source of satisfaction and knowledge to me. I think work of this kind should be always conducted with the clinical worker concerned. As things are at the hospital at present that is not always possible, so that the clinician does not benefit very much, except when he makes inquiries about his case or it is brought up at a staff meeting. I should like to share the undoubted benefit that I receive and to stimulate, moreover, a study of the available literature while the case in all its bearings is fresh in the memory. At present I am the only one who gets much out of it all, and this contribution tonight is an attempt to share the luck. It has seemed to resolve itself into an *exposé* of my colleagues' mistakes. Perhaps I should promise to tell you all about my own and in that way escape a present confession of many failures.

Reports of Cases.

A CASE OF CONGENITAL MALARIA.

By EVA A. SHIPTON, M.B., B.Sc. (Sydney),

AND

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BABY T.M., male, aged ten weeks, was referred to one of us (D.V.) by a colleague for investigation on January 18, 1935.

The history given by the medical attendant was that he had first seen the baby four days previously at 10 p.m. The baby was then slightly cyanosed, appeared collapsed and had an extremely rapid heart rate; the temperature was 38·9° C. (102° F.). The mother stated that the baby had previously had many similar attacks, commencing on December 25, 1934, and occurring first on alternate days, but recently every evening between 8 and 10 o'clock.

The baby at the beginning of these attacks was pale, grey and cold; later it became clammy and hot. In between these attacks the child looked pale, was somewhat irritable and was having five to six greenish motions per day. The temperature generally was subnormal. Further questioning of the mother revealed the fact that the baby was jaundiced at birth and for a month afterwards; the jaundiced appearance gradually changed to extreme pallor.

On examination the child was extremely pale, the mucous membranes appearing bloodless. The pulse rate was 130 per minute. The temperature was 36·2° C. (97·2° F.). The heart sounds lacked tone. The only positive finding, in addition to the pallor, was a large spleen, very easily palpated below the costal margin. The mother stated that she had returned from the Solomon Islands two months prior to the birth of the child, that she had suffered from malaria for two years and that she had had a rigor forty-eight hours after the birth of the child.

The following is the result of the blood count on the baby on January 18, 1935. The red blood corpuscles numbered 1,670,000 per cubic millimetre; the hæmoglobin value was 31%, the colour index was 0·9. Pronounced anisocytosis was present with many small forms and some slightly larger than normal, but no macrocytes. Poikilocytes were present. Numerous polychromatic forms were seen. Reticulocytes numbered 12%. Two normoblasts were seen while 200 leucocytes were being counted.

The parasites of benign tertian malaria were very numerous. All stages were seen in the films and some cells contained two ring forms.

Leucocytes numbered 5,600 per cubic millimetre. Of these, the neutrophile cells were 36%, lymphocytes were 53% and monocytes 11%.

No plasma cells were seen. Some of the monocytes showed vacuoles in the cytoplasm, but none containing pigment were seen. The lymphocytes and neutrophile cells were all normal cells. To the Van den Bergh test a direct negative and an indirect positive reaction were obtained.

There was reported a severe anæmia of the hæmolytic type. The whole blood picture was accounted for by the malarial infection.

The child was treated with "Euquinine" immediately and, as evidenced by the temperature chart and blood count, made an uninterrupted recovery.

An examination of the blood on January 29, 1935, showed 3,540,000 red cells per cubic millimetre and a hæmoglobin value of 57%. No parasites could be found.

The parasites of benign tertian malaria were found in the mother's blood on March 1, 1935.

Comment.

This case raises once more the question of the proof of congenital malaria.

The literature as far back as 1858 contains examples, indirect proof and more or less direct proof of the transmission of malarial infection from the mother to the child, so that at present it is generally believed that a baby may be born harbouring the parasite in its tissues.

Stein[1] reports the case of an infant born in New York which was well until two and a half months of age, but which died of quartan malaria one month later. The mother had lived in Palestine in a district infested with malaria, but to her knowledge had not suffered from the disease. She had no splenic enlargement and parasites were not found in her blood.

Schadow[2] reviews the literature and reports a case of benign tertian malaria in a male infant whose mother had been treated by malaria for cerebral syphilis. Owing to the bad psychical state of the mother, the child was removed from contact with her immediately after birth. At the beginning of the third week the child became feverish and anæmic and the parasites were demonstrated in his blood. Recovery ensued after treatment with quinine and blood transfusions.

Manson[3] states that: "Malarial parasites have been demonstrated in the blood of a child before birth, while Heiser has recorded the case of an infant seven days old with crescents of *Plasmodium falciparum* in its blood."

These examples, together with the one here presented, that is, a child born in a suburb of Sydney, New South Wales, entirely free from primary malaria, and born to a mother who had a history of malaria for two years previously and who actually had a rigor two days after the child's birth, is strong evidence that the child was suffering from congenital and not acquired malaria. The child had been protected since birth by mosquito netting while asleep.

In favour of the case being one of acquired malaria is the fact that the anopheline mosquito is known to be present in Sydney and that at least one alleged case of acquired malaria has been reported.[4] Also the first pale sweating turn noticed by the mother was when the child was six weeks old. This just allows for the possibility of the infection being transmitted from the mother to the child by a mosquito.

However, Schadow[2] notes that both in his case and the one reported by Leven there is a latent interval after birth before the appearance of symptoms, and it is possible that in the case here reported the child had milder symptoms earlier than those noticed at the age of six weeks, which were not severe enough to excite the attention of the mother.

This report fits in with other reports on congenital malaria, on the basis of circumstantial, but nevertheless incontrovertible, evidence.

Acknowledgement.

We wish to thank Dr. W. L. Calov for advice in regard to the treatment of this patient.

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[1] M. L. Stein: *American Journal of Diseases of Children*, Volume XLVI, December, 1933, page 1467.
[2] H. Schadow: *"Impfmalaria als Ursache kongenitaler Malaria"*, *Münchener Medizinische Wochenschrift*, Volume LXXVIII, June 5, 1931, page 947.
[3] Manson: "Tropical Diseases", 1929, page 22.
[4] R. A. Money: "A Case of Malaria Acquired in Sydney", THE MEDICAL JOURNAL OF AUSTRALIA, August 28, 1926, page 283.

Reviews.

CHRONIC RHEUMATISM.

OSLER expressed the opinion that there was truly no such a state as chronic rheumatism. Perhaps he wished to make clear the distinction between rheumatic fever, with all its cardiac dangers, and the various forms of arthritis, the activities of which centre round the joints. However, though the term is probably unscientific, most physicians will understand that a group of serious diseases is included when we speak of chronic rheumatism, and that we admit a certain family resemblance between members of the group. R. Fortescue Fox and J. Van Breemen have written a very interesting and comprehensive book on the subject, and not only shed light upon it, but also put in some special pleading for the campaign against rheumatism.[1] Particular attention is paid to the functions of the skin and its disturbances, a point of special interest in relation both to climate and to physical therapy. In fact in discussing ætiology they give very much more space to the cutaneous system and to external factors than to constitution and focal infection. The descriptions of the clinical pictures are clearly given, as is also the account of treatment, which is discussed from the standpoints of objects and methods. Nearly one-quarter of the book is concerned with rheumatism as a problem of public health and economics, but nevertheless the sections dealing with the handling of the individual patient are detailed and practical. In Australia one cannot help feeling how inadequate are the facilities available for treatment of the rheumatic diseases, and although the economic ravages due to loss of work and disablement are much less than in Europe, one wonders how long it will be before any concerted move will be made here to gather information and provide treatment. From the points of view of imparting information as to the cause of rheumatism and how to undertake the treatment of patients, and also of the propaganda so needed here against this important disease the book may be warmly commended.

More modest is the book of G. D. Kersley, who has written for the general practitioner.[2] He has omitted perhaps rather too much in the endeavour to provide a succinct account of rheumatic disease; perhaps the book would be better if an attempt were made to deal with rheumatic fever. The detail in matters of treatment is rather curtailed, but the book is clear and brief and gives a reliable description of the various types of joint disturbance with their pathology, and summarizes the different forms of treatment. Anyone requiring a brief guide to rheumatic disease can rely on the information given, and can obtain the rudiments of the subject without padding.

Both books are clearly printed, and the former is in the style already familiar in the "Recent Advances" series, with many useful illustrations.

[1] "Chronic Rheumatism, Causation and Treatment", by R. F. Fox, M.D., F.R.C.P., F.R.Met.S., and J. Van Breemen, D.M.; 1934. London: J. and A. Churchill. Demy 8vo., pp. 371, with eight plates and 38 text figures. Price: 12s. 6d. net.

[2] "The Rheumatic Diseases: A Concise Manual for the Practitioner", by G. D. Kersley, M.A., M.D., M.R.C.P., with a preface by F. R. Fraser, M.A., M.D., F.R.C.P.; 1934. London: William Heinemann (Medical Books) Limited. Demy 8vo., pp. 104, with illustrations. Price: 6s. net.

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MOTOR TRAFFIC ACCIDENTS.

MOTOR TRAFFIC, one of the blessings of the modern age, is also a menace to human life. Almost every day the newspapers report the occurrence of motor car accidents, and on each Monday morning they almost invariably record deaths caused by motor cars during the week-end. The occasional motorist emerges at the end of the week, and may be relied upon to appear with unfailing regularity among the casualties reported on Monday. Admittedly the number of motor cars on the roads is constantly increasing, but this is not sufficient explanation for the number of deaths that occur. During 1933 the number of deaths caused by automobiles in the Commonwealth of Australia was 777; of the persons killed 614 were males and 163 were females. *The Sydney Morning Herald*, commenting on this subject, has drawn attention to the extraordinary complacence with which motor car accidents are accepted as a feature of modern life. It refers to an English paper as having stated that too much was being said about the accident rate, and that it was no worse than that of accidents in factories. *The Sydney Morning Herald* points out that if some mysterious disease were working any such havoc as reckless motoring is doing throughout the whole of the mechanized world, there would be an overwhelming outcry and an insistent demand for investigation and remedy. The preservation of life is the concern of the medical profession. The reference of the English paper to industrial accidents is apt. The medical profession, through its activities in industrial hygiene, makes efforts to eliminate or at least to reduce the number of accidents in factories; it should also pay some attention to the motor accident problem.

Under existing regulations almost any person over a certain age can procure a motor driver's licence. There is no difficulty—a simple driving test has to be passed and no stringent investigations into the applicant's fitness are made; nor is his intelligence considered. Once a licence is issued, it is renewed every year without question. The bearer may have been involved in any number of accidents and no official notice will, as a rule, be taken by the authorities. Endorsement will be made on the back of the licence of any convictions for breaches of the traffic regulations, but these will be the only stain on the motorist's character. Only for serious breaches or for gross carelessness resulting in injury to or death of another person will the licence be suspended, and even in these circumstances suspension will generally be for a limited period only. That some persons are unusually liable to meet with accident has recently been shown by the Medical Research Council of Great Britain. The Industrial Research Board of the Council has found that accident proneness in factories is subject to definite laws.

Not only are some people more prone to accidents than others exposed to the same risks, but those specially prone to accidents sustain an undue number of accidents both in the factory and out of it, and those who sustain an undue number of one kind of accident also sustain an undue number of other kinds. Accident proneness is a relatively stable quality, so that those who sustain an undue number of accidents in one period of exposure will tend to do so in other periods of exposure. Further, those who sustain an undue number of minor accidents also tend to sustain an undue number of major accidents. Thus it follows, and is in fact found, that if those who have an undue number of accidents in their first year of exposure are eliminated, the subsequent accident rate of the group is diminished.

The Council states that practical application of the principle is now frequently made in industrial occupations and that the number of accidents can be materially reduced by detecting and removing the accident-prone. An investigation is now in progress dealing with motor accidents. From a statistical examination of the road accident records of four groups of omnibus and private drivers, totalling 2,604 persons, it has been shown that in each group certain individuals are more liable to accidents than others. This holds true for all accidents, including those for which the driver was not held to blame. From a more intensive study of 179 motor drivers from among the group of 2,604 persons it was concluded (the findings are stated as preliminary results) that the findings in regard to motor accidents are similar to those previously obtained with regard to factory accidents.

The subject of accident proneness is one of the greatest interest; its psychological basis must be left for the discussion of readers. The Medical Research Council sees two ways of preventing the accident-prone from engaging in specially dangerous occupations. The first is by the use of appropriate tests that will determine beforehand who are most likely to sustain accidents. The second is by removing those who in an initial period of exposure sustain an undue number of accidents. Of course, pronenesss to accident may be only one factor in the causation of an accident; it may not be operative at all. If worth-while results are to be achieved, definite data as to accident causation must be forthcoming. These data could be obtained by setting up such an official body as a Motor Accident Investigation Board. This body would need to operate over a period of years, and it would be necessary for it to investigate accidents of every kind and of every degree of severity. Since the vast majority of accidents come within the purview of insurance companies, it would be necessary for the obligation to be placed on insurance companies of reporting all motor car accidents to the board. The board would investigate not only the proneness of the driver to accident, but also such aspects as the construction of the motor car and the peculiarities of roads. For example, it might be found that accidents were liable to occur with some of the so-called improvements in the newer types of motor car, except when they were driven by the most experienced drivers. Again, it might be found that road gradients and/or camber of a particular type were frequently associated with accident. It would probably be found, however, that the most variable factor of all—the human mind and its power of concentration on the work of driving—was generally at fault. After a period legislation suitable to the findings of the board would have to be passed by the legislature. Something of this kind will have to be done before long, for the toll of the roads in human life is becoming larger every year. It should be remembered also that if the deaths from motor car accidents are reduced in number, the number of injuries not resulting in death, with all their unnecessary pain and disability, will also be reduced.

Current Comment.

THE PROGNOSIS OF LIPOID NEPHROSIS.

FOR a number of years the literature of medicine has contained many contributions to the subject of nephrosis, though this supposedly degenerative and not actually inflammatory lesion of the kidneys is distinctly uncommon. But it is very important on account of its relation to the problems of renal physiology and pathology. It is of interest to consider what are the most striking features of the nephrotic syndrome; they are albuminuria (or, as it actually is, proteinuria) and œdema. Now the test for the presence of albumin in the urine is probably the commonest of all chemical tests used in the practice of medicine, and every practitioner invariably looks for the evidences of œdema when he has any suspicion of renal inadequacy. Any observations bearing on the causation or importance of these signs are therefore of interest to all.

Herman Schwarz and Jerome L. Kohn have followed up thirty-six children in the service of the Mount Sinai Hospital in New York, all of whom have been treated for a renal affection considered by them to be lipoid nephrosis.[1] They set down the criteria of this syndrome as an insidious onset with the appearance of an œdema that is subject to definite remissions, the presence in the urine of large amounts of albumin with very few formed elements, a normal level of urea in the blood, but increase in the blood cholesterol, decrease in the

[1] *American Journal of Diseases of Children*, March, 1935.

serum protein and normal blood pressure. A good follow-up system has enabled the hospital to keep in touch with the patients over periods of from two to ten years. The patients were grouped as recovered when they had been free from œdema and maintained a normal urinary and blood chemical state for one year; of these there were nine, that is, one-quarter of the total number investigated. Of the others, eighteen died, and the remainder seemed well, but still were passing albumin in the urine. That the condition is far from benign will be seen by the high mortality rate of 50%. The children who recovered belonged to two classes, those who had been affected only for a short time, in whom the process was apparently mild, and those who had clinical symptoms for well over a year. Some of these little patients were very young, for age seems to have little bearing on the prospect of recovery. In the children who had suffered for longer periods there were more cases of intercurrent infection, but it is cheering to realize that even severe infections, such as purulent peritonitis, did not prevent recovery in some instances. Also, infections occurring when the child was clinically well did not necessarily revive the kidney disease. One case is instructive. A girl of eight years was treated in hospital for one year for a persistent nephrosis. On discharge she was still passing albumin in the urine, but œdema had disappeared, and the serum protein was normal. The albuminuria persisted for four years, and then for three years the urine remained normal, but during a routine examination after this period albumin was again found in the urine, though there were no formed elements. The systolic blood pressure was 140 millimetres of mercury and the diastolic 95 millimetres, the blood urea value was 21 milligrammes per 100 cubic centimetres, and the urinary concentration test gave a normal figure. Several very interesting observations may be made about this case. Incidentally, the authors are unduly optimistic in speaking of cure in such cases; they ask if a contracted nephrotic kidney or some new condition is developing, but surely most clinicians would suspect that chronic renal changes are insidiously taking place here. Again, the long period of remission should be noted; this strongly resembles what is known to happen in true nephritis of the latent type. But the most significant point is that the crucial observation was the finding of protein in the urine, a form of examination universally carried out, as remarked before. It cannot be too strongly stressed, therefore, that no practitioner can afford to treat lightly the phenomenon of albuminuria, for with all that has been written and talked about the chemical investigation of the urine and the blood, the simple bedside test of boiling the urine and adding acid is still one of the most significant.

Of the children whose condition was found to be improved, but who could not be regarded as quite well, one is also interesting. This child suffered from a prolonged osteomyelitis, and it was thought for some time that the renal disturbance was of the amyloid type, but after observation extending over several years the authors think he now has a progressive glomerular nephritis. Once again the sinister possibilities of what may appear to be a purely tubular degenerative change should be noted. Indeed, it appears to be extremely doubtful if some glomerular changes are not present in practically all cases. The children who died succumbed to peritonitis in most instances, and to some infection in every case, and *post mortem* examination revealed that while in the cases of briefer duration the changes in the kidneys were of variable degree, and chiefly of the nature of tubular degeneration, in those of longer standing there was usually damage of the glomerular apparatus together with some interstitial change.

The authors remark that the course of a true subacute glomerular nephritis is quite different from that of lipoid nephrosis, the former being notoriously lethal, and that recovery from the latter depends on the capacity of the patient to withstand severe secondary infections. They admit, however, that the occasional demonstration of true inflammatory sequels in the kidney indicates latent possibilities in the syndrome. The most practical points to be remembered are that infections are very dangerous to these children (and in general these are not preventible), and that it is vitally important that the urine be regularly examined over long periods.

ANTIVENENE FOR SPIDER BITE.

IN *The Canadian Medical Association Journal* of January, 1935, there appears a reprint of "Science News Letter", 1934, 26, page 339, concerning an antivenom serum for the bite of the black widow spider (*Latrodectus mactans*). The female is much larger and far more deadly than the male, which she kills soon after the breeding season, hence the popular name. The species is also termed the "hour-glass spider," from the presence on the lower part of the abdomen of two red triangular spots with the points touching, thus giving a rough outline of an hour-glass. This species is common in the United States of America and has recently appeared in the southern part of British Columbia. Weight for weight, the venom is more poisonous than that of the rattlesnake. At one time the spiders were found only in rural districts, but of recent years they have become urban. Like the Australian species, they are timid and do not attack unless molested. The bite itself is not painful, but, within an hour or so after infliction of the bite, a numbing pain ascends the bitten extremity and becomes localized in the abdominal muscles, back and chest, with spasms and intense suffering. Difficulty in breathing results from contraction of the chest muscles. Other manifestations are high fever, nausea, vomiting and unconsciousness. Similar results may follow bites of the Australian species. A convalescent serum has been employed in treatment, but Professor F. D'Amour, of the Denver University, does not consider it satisfactory. D'Amour has perfected a serum which is believed by him to be the first highly

potent antivenom against bites by these spiders. This serum was obtained from the blood of rats which had been given small injections of venom removed from the spiders' glands. Its efficacy was first demonstrated on a vineyard worker. The serum was not administered until three hours after infliction of the bite, yet relief was immediate. D'Amour's interest was first aroused on hearing of the work of Dr. Allan Blair, of the Alabama University. This enthusiastic investigator, as reported in a recent issue of this journal, permitted himself to be bitten by a large specimen of the spider and endured severe pain for hours in order to allow scientific friends to observe and record the symptoms. This spider is increasing at an alarming rate throughout the United States of America, and it is feared that it will become a greater menace than the rattlesnake. Several deaths have been reported in the United States within the past two years. It is claimed that the new serum gives prompt relief if given as long as three and a half hours after infliction of the bite. This is of practical importance, as it often happens that bitten people are at a distance from medical aid.

The Australian species (*Latrodectus hasseltii* Thorell) is popularly termed the "jockey spider" or "red-back". It is also known as Katipo in New Zealand. Its habits are similar to those of the United States species, and the symptomatology of its bite also resembles that of the American congener. W. W. Ingram and A. Musgrave have described fully in THE MEDICAL JOURNAL OF AUSTRALIA[1] the habits and toxic effects following the bites of this and other poisonous Australian spiders. C. H. Kellaway[2] has studied the poisonous effects experimentally. So far it has not been deemed necessary to manufacture an antiserum for bites of this spider in Australia. Were many deaths reported, this would doubtless be done.

"PLASMOQUIN" AND "ATEBRIN".

THE synthetic anti-malarial drugs "Plasmoquin" and "Atebrin" have been the subject of much investigation; yet many medical practitioners seem to be ignorant of the most important known facts concerning them and the dangers that are known or suspected. After the great boom of its early years, "Plasmoquin" has rightly found its place in the limited sphere of an anti-gametocyte in malignant malaria; yet some medical practitioners still employ it in what are now known to be toxic doses, both as a therapeutic and a prophylactic. Although it has been proved that there is no *therapia sterilisans magna* in malaria, patients are still told by medical practitioners that a course of "Atebrin" (1·5 grammes over a period of five days) will completely eradicate their malaria. It is no great rarity for a patient to be attacked with malaria within a day or so after taking his last dose of "Atebrin"; this is not to be wondered at, in view of the findings of the Malaria Commission of the League of Nations (1933). "Atebrin" is a far more useful drug than "Plasmoquin", and in many cases can be employed with advantage in place of quinine; unfortunately its value is liable to be discredited by its failure to satisfy the extravagant demands on its powers. "Atebrin" often produces a yellowish pigmentation of the skin. This is not due to disturbance of liver function, as many have supposed, but to staining of the tissues with the drug itself. It is of no serious significance; but it probably indicates that the drug is not being eliminated as rapidly as when no staining occurs. This is important, as the presence or absence of staining may serve as an indication of whether or not much "Atebrin" remains in the system. Certain toxic effects of "Atebrin" have been reported; they are: abdominal pain, headache, anorexia, hæmoglobinuria, mental depression or excitement, and even psychosis. These toxic effects seem to be nearly always temporary. It is doubtful whether they should all be attributed to "Atebrin"; close investigation reveals that in all cases of severe poisoning "Plasmoquin" has been given in conjunction with "Atebrin". Possibly the severe symptoms are produced by the "Plasmoquin"; possibly the toxic effects of either drug are enhanced by the presence of the other. A series of three interesting papers, appearing in a recent issue of *The Indian Medical Gazette*, help to increase the knowledge of the toxicity and therapeutic and prophylactic value of these drugs.

In the first paper, R. N. Chopra and R. N. Chaudhuri discuss the toxicity of "Plasmoquin" and "Atebrin".[1] They remark that there appears to be a tendency for medical practitioners to prescribe quinine and "Atebrin" together. Both these drugs have their effects mainly on the asexual forms of the parasite, and the effects do not appear to be any greater when the two are used in combination. A combination of "Plasmoquin" (which has a destructive effect on the sexual forms) and "Atebrin" would appear to be more rational; it has been used freely in India. They remark, however, that while this combination "may be theoretically sound and the most effective in the treatment and eradication of malaria, it appears to be toxic". They suggest that the presence of "Atebrin" increases the toxicity of the "Plasmoquin". They report eleven cases in support of this view. In one case the patient took 0·3 gramme of "Atebrin" and 0·03 gramme of "Plasmoquin" every day for five days, then, without taking more, became affected with tachycardia, substernal and epigastric pain, and cyanosis. "Plasmoquin" in a dose of this size rarely causes any toxic symptoms. Chopra and Chaudhuri stress that the main function of "Plasmoquin" is the destruction of the sexual parasites, hence prevention of the dissemination of the disease by mosquitoes; treatment for two or three days with 0·02 gramme of "Plasmoquin" a day is all that is required in India to cause disappearance of crescents from the peripheral blood. They further

[1] THE MEDICAL JOURNAL OF AUSTRALIA, July 1, 1935, page 10.
[2] *Proceedings of the Australasian Medical Congress (British Medical Association), Third Session*, Sydney, 1929, page 227.

[1] *The Indian Medical Gazette*, January, 1935.

assert that patients should not be allowed to use these drugs except under direct medical supervision. With this view we concur. Chopra and Chaudhuri might have mentioned that "Plasmoquin" is required only in subtertian malaria, as in benign tertian and quartan, quinine or "Atebrin" alone suffices to eradicate the gametocytes.

In the second paper, C. D. Newman and B. S. Chalam discuss the use of "Atebrin" and "Plasmoquin" in treatment.[1] They gave as a routine procedure either the two drugs together or "Atebrin" for five days, followed by "Plasmoquin" for five days. The dosage they employed was 0·3 gramme of "Atebrin" and 0·03 gramme of "Plasmoquin" a day; proportionately smaller doses were given to children. Untoward symptoms occurred in 5·8% of cases when the drugs were given consecutively, and in 21·05% when they were given simultaneously. The toxic symptoms were more severe when the drugs were given together. They give both drugs in every case, whatever the type of parasite, because, first, the diagnosis has often to be left to "medical subordinates", who cannot always be relied on to identify the parasite, and, secondly, they believe the relapse rate in benign tertian malaria is lower for some reason or other when "Plasmoquin" is given as well as "Atebrin". The most interesting feature is the low incidence of recurrence of malaria; of 334 patients treated, only 50 suffered a recurrence within twelve months. It is probable that some at least of these had acquired fresh infections. The inference is that for some reason unknown "Atebrin" has a more pronounced effect than quinine in the prevention of relapse. It would seem that the great majority of the patients in this series suffered from subtertian malaria. This should be borne in mind when an evaluation of the results is being made; for subtertian malaria has no great tendency to relapse, and "Atebrin" is known to be more effective against it than quinine. Although Newman and Chalam conclude that treatment with "Atebrin" and "Plasmoquin" is a distinct advance, we remain sceptical concerning the value of "Plasmoquin" as anything but an anti-gametocyte in subtertian malaria.

In the third paper, D. P. Williams and Rasamay Bhattacharyya discuss their experience with "Atebrin" and "Plasmoquin" in treatment and prophylaxis.[1] The most important point brought out by them is the necessity, in estimating the value of a drug in the prevention of relapse, of making a distinction between children, who are highly susceptible to malaria, and adolescents and adults, who have had time to acquire some degree of immunity. They state that the administration of "Atebrin" followed by "Plasmoquin" fails "to fulfil the somewhat exaggerated claims made on their behalf". Unfortunately they used slightly smaller doses than the optimum, excepting when patients were febrile, and also their experiment was carried out in a highly malarious locality, where reinfection was always likely.

[1] *The Indian Medical Gazette*, January, 1935.

It is clear that "Atebrin" is a valuable drug; it is particularly useful in subtertian malaria. It can be given without fear of serious toxic effects, providing it is given alone; but it is not suitable for prolonged administration. "Plasmoquin" has a limited sphere of value, to which it should be confined; its greatest service was to show that the synthesis of an anti-malarial drug was possible. Let it retain the dignity of a pioneer and let it not be credited with mysterious powers to its ultimate discredit.

PROLONGED ARTIFICIAL RESPIRATION.

SOME time ago reference was made in these pages to a most remarkable case in which artificial respiration was continued, first by manual means, and subsequently by mechanical devices, for a period of two years. This, together with such publicity as is given from time to time to the use of the Drinker apparatus, reminds us that the necessity for such sustained methods of resuscitation sometimes arises. An apparatus has recently been described, designed by R. W. Paul, to apply the method of Sir William Bragg employed in the case mentioned.[1] This is a pulsator which inflates an air-bag encircling the thorax of the patient below the axillæ, causing a rhythmic increase in air pressure, which is measured by a manometer. This causes a correspondingly rhythmic compression of the thorax at a rate that may be varied from 15 to 30 times per minute. The pressure used is about 30 to 40 millimetres of mercury for an adult, and when the pressure is adjusted to the desired maximum the device will secure a steady series of pulsations of the required force and frequency for long periods without attention. Power may be obtained either from the local electrical supply mains, the necessary motor using a sufficiently small ampèrage to permit connecting it up to an ordinary lamp socket, or from the water supply, according as electric or hydraulic drive is desired. In each case the motor drives a stout bellows which supplies the air pressure to the bag. The chief point of interest about this apparatus is that it is readily portable, a feature that adds very considerably to its general utility; another advantage is that the machine, though relatively small and quiet, need not be placed near the patient, as the flexible hose connexion can be of some length.

Investigation of a normal subject under a fluorescent screen demonstrated that the diaphragm was raised, the antero-posterior diameter of the thorax was increased, and the lateral diameter was decreased while the machine was working. The expiratory phase of the cycle of respiration was exaggerated, but not more than would be produced by forced breathing; this was with a pressure of 47 millimetres of mercury, which did not give rise to any discomfort to the subject. It would be interesting to have further details of the use of this Bragg-Paul pulsator, as it would appear to be a useful type of apparatus without being too elaborate.

[1] *Proceedings of the Royal Society of Medicine*, February, 1935.

Abstracts from Current Medical Literature.

GYNÆCOLOGY.

Vesico-Vaginal Fistula.

D. FONSECA (*Journal d'Urologie*, August, 1934) proposes the use of distension of the bladder in order to open up the folds of the vesico-vaginal septum during operations for the cure of vesico-vaginal fistula by the vaginal route. This distension is effected by introducing a child's toy balloon into the bladder through the urethra and then distending it with water. Not only is the field of operation better exposed by this ingenious plan, but, in addition, the surgical manœuvres are facilitated by the fact that the edges of the fistula rest on a supporting bed.

Primary Carcinoma of the Fallopian Tube.

W. WASSLOW (*Monatsschrift für Geburtshülfe und Gynäkologie*, December, 1934) discusses the ætiology of primary tubal carcinoma. Many authorities presuppose a chronic inflammatory state because gonococci have frequently been found in the tubal fluid in cases of carcinoma. In both of the author's two cases signs of old pelvic inflammation were present. The condition generally occurs about the menopause and possesses few characteristics. There may be menorrhagia or metrorrhagia, pain which tends to radiate down the thighs, and a sero-purulent blood-stained discharge. The forms of carcinoma noted were papillary, alveolar and, more commonly, a mixture of both. Owing to the difficulty of preoperative diagnosis, the prognosis is not good—nine patients recovered in a published series of 314. Metastases to the lungs are common. The best treatment is radical removal of the uterus and appendages with clearance of the lymphatic system as far as possible. In some cases salpingectomy alone was followed by recovery, but this treatment is not advised as a routine measure.

Radiation Treatment of Carcinoma of the Cervix.

WILLIAM P. HEALY (*The American Journal of Roentgenology and Radium Therapy*, November, 1934) reports a new series of 26 cases in which X ray technique has been altered in order to increase the amount of effective radiation given to the pelvic field with the idea of controlling carcinoma of the cervix for a longer period in advanced cases and to raise the percentage of permanent cures. Since 1929 X rays are always given first, except in the very earliest cases; the cancer field is thereby rendered more suitable for radium application. Dosage to each field has been raised from 700 *r* to 2,000 or 2,400 *r* in 200 *r* doses twice a day, the course being completed in twenty to thirty days. Target distance is raised from 50 to 70 centimetres. As soon as the X ray treatment is finished, radium is applied in the vaginal canal against the cervical lesion for about 1,500 millicurie-hours, filtered through two millimetres of brass. Immediately following this two radon capsules are placed in the cervical and uterine canals for 3,000 millicurie-hours, filtered through 0·5 millimetre of gold and two millimetres of black rubber. Biopsies are taken at intervals of three to five days during the course of treatment; these biopsies verify the clinical evidence of regression and indicate that permanent healing cannot be expected with the external irradiation alone. No intestinal complications were noted with this dosage, nor any severe local reactions in the form of cellulitis, nor hæmorrhage. There was little or no bladder or rectal distress; the skin reactions were rarely severe, although 0·5 millimetre of copper and 2·0 millimetres of aluminium only were used as filters.

Actinomycosis of the Female Genital Tract.

E. JUNGHANS (*Monatsschrift für Geburtshülfe und Gynäkologie*, December, 1934) gives the details of a case of primary actinomycosis of the genital tract. Following upon a septic abortion the patient complained of pain in the vesical region. Vaginal examination revealed a painful mass, which was diagnosed as parametritis, bilateral and anterior. X ray therapy was given, and as the general condition did not improve, curettage was performed. The scrapings were abundant and showed typical actinomycotic bodies. When the speculum was inserted, a laceration in the anterior vaginal wall was observed and was considered to be the portal of entry. The patient rapidly became worse and died. Autopsy revealed massive infection of the pelvis with metastases in the abdomen and liver.

The Treatment of Genital Prolapse.

W. FLETCHER SHAW (*The Journal of Obstetrics and Gynæcology of the British Empire*, December, 1934) describes the structure of the pelvic floor and enumerates the cause of prolapse, and describes the type of operation which has been continuously performed at Manchester since 1888, when Archibald Donald first carried it out. At a later date Fothergill wrote extensively about the subject. The operation advised by the author is one slightly altered from the original operation of Donald, but the main principle is the same. The operation is described in detail, and the results of the seven-year period 1924-1930 have been followed up. Of the 664 patients, 640 were completely cured, that is, 96%; in 24 patients, or 3%, the result was not satisfactory; five patients had subsequently borne children; five failed to come for further investigation and were regarded as failures. During this period there was one fatal case, and during the author's twenty-seven years' experience of this operation, in which 2,293 operations have been performed, the mortality rate is 0·43%. Included in this series of patients are 211 who are over fifty years of age, and of these 97% were cured. The author is of the opinion that genital prolapse can be cured by the operation of colporrhaphy. In the majority of cases anterior and posterior colporrhaphy are combined with amputation of the cervix, but each case is treated on its own merits, and the amount of tissue removed and the type of suture used depend on the situation and the maximal laxity. The operation does not cause trouble in subsequent labours and is equally beneficial in old age and in youth.

Lipiodol Injection and Air Insufflation in Sterility.

B. RABBINER (*American Journal of Obstetrics and Gynecology*, January, 1935) quotes the literature and his own experience with the use of lipiodol injections. His attention was called to a patient injected with lipiodol who left the surgery and on returning home was seized with pain and died a few days later with fulminating peritonitis. Another case is quoted in which lipiodol was retained in a bicornuate uterus for twenty-two months. The author comes to the conclusion that lipiodol injections are not harmless; they carry a morbidity and mortality, even though small. In sterility, in cases in which one tube is occluded or both tubes show partial occlusion, lipiodol injections should be used cautiously, as complete occlusion may result. The contention that lipiodol may remain in the peritoneal cavity for one year or more and result in serious pathological change has been verified by the author's personal experience and publications of others. Misinterpretation is not unlikely in the reading of the salpingogram by the inexperienced, and errors are few after transuterine air insufflation.

OBSTETRICS.

Hookworm Disease as a Complication of Pregnancy.

G. A. W. WICKRAMASURIYA (*The Journal of Obstetrics and Gynæcology of the British Empire*, April, 1935) considers that the greatest danger of ankylostomiasis appears to be when it occurs as a complication of pregnancy. As a result of investigation carried out at the De Soysa Lying-in Home, Colombo, which is the premier maternity hospital in Ceylon, he has come to the conclusion that it is one of the gravest complications of pregnancy. It very commonly causes abortion, miscarriage, premature birth and stillbirth, particularly in untreated cases. Of patients suffering from hookworm, 90% exhibit albumin-

uria and anasarca in the second half of pregnancy, and in the vast majority of patients studied definite renal function has been present. Intrauterine fœtal death is far commoner in this condition than in eclampsia and preeclampsia. The cause seems to be placental insufficiency resulting from placental disease, retroplacental clots, premature separation, or an unduly small placenta. Actual labour is generally easy, owing to the smallness of the infant. However, in the puerperium, owing to the low vitality, infants are prone to develop various complications. The influence of pregnancy on hookworm disease is a grave one in the majority of patients. General anasarca and dropsical conditions of the vulva appear to be the rule. Not only does pregnancy cause a general exacerbation of the disease, but it also gives rise to a greatly increased mortality. The influence of labour resolves itself almost entirely to the effects of labour on the heart, which in most cases is dilated. The largest number of patients die in the puerperium, either from cardiac failure or, less commonly, from *post partum* shock. Should the patient survive labour in the early days of the puerperium, it is not uncommon to see a great amelioration in general health take place. The tragedy is that poverty may compel the patient to live in the same unhealthy surroundings, with the result that reinfestation takes place, which further retards the recuperative process and eventually causes her death.

Traction in Forceps Deliveries.

B. WYLIE (*American Journal of Obstetrics and Gynecology*, March, 1935) determined by means of a traction handle with a spring mechanism inserted, the exact amount of force required to effect delivery under various conditions. The pull was for the duration of the uterine traction only, and an effort was made never to exceed the least amount of traction needed to advance the head. The traction observation was made in 880 cases. These were unselected, with the exception of thirteen cases that required the use of Tarnier blades and hence were not included. The most extreme variation was encountered between individual cases, not only in the force, but in the number of pulls required. For example, among the *primiparæ* the range was from a single pull of five kilograms to a series such as 15-14-18-19-21-23-21-21-13-12, or, in another case, 26-27-27-30-29-28-23-12. Amongst the *multiparæ* the traction required was also variable. The maximum traction recorded in any one single pull was 34 kilograms. The maximum number of pulls in any single case was ten. The average pull in 321 *primiparæ* was 15·45 kilograms, or 34·0 pounds, and the average number of pulls was 3·7. The authors have estimated a "resistance index" which they take to be the product of the average individual pull multiplied by the average number of pulls in any series, and for *primiparæ* this was 57·2. In *multiparæ*, of which there were 559, the average pull was 11·15 kilograms (24·5 pounds), the average number of pulls was 1·9, and the "resistance index" was 21·2.

Acute Mastitis of the Puerperium.

A. A. MOON AND B. GILBERT (*The Journal of Obstetrics and Gynæcology of the British Empire*, April, 1935) record their findings in one hundred consecutive cases of acute puerperal mastitis in the City of London Maternity Hospital. As regards ætiology of the condition, it was found that although age played no part, yet it was more common in the *primiparæ*. It was noticed that there was an absence of puerperal mastitis in the district patients compared with the patients in the hospital, which led them to believe that already existing infective foci may not be the important predisposing factor often suggested. Hæmorrhage and albuminuria did not have any relation to the development of mastitis. Interference with normal labour by induction and Cæsarean section is of considerable importance. The authors found that the onset of pyrexia is by far the most common in the second week. Unilateral mastitis is twice as common as bilateral. Only one-quarter of cases of acute mastitis resolved. The authors are in favour of delayed operation after waiting localization. The presence of cracked nipples, in their opinion, tends to be exaggerated as an ætiological factor. Three different methods of local prophylactic nipple treatment failed to alter the incidence of mastitis. *Staphylococcus aureus* was the cause of all the breast abscesses examined in this series. Among the infants of these patients there was a mortality of 3% and a morbidity of 8% due to intestinal infection, presumably from the mother's milk. The authors are of the opinion that the baby should be removed from both breasts as soon as a definite diagnosis of acute mastitis is made. The bacteriological investigations show that the presence of *Staphylococcus aureus* alone does not necessarily mean an acute mastitis, other factors being necessary, as a virulent strain and lack of immunity.

Blood Chemistry in Preeclampsia and Eclampsia.

H. J. STANDER AND J. F. CADDON (*American Journal of Obstetrics and Gynecology*, December, 1934) give a detailed report of the blood constituents in 108 eclamptics and 40 preeclamptics. The authors found that, although the investigations were first started in an endeavour to determine the cause of eclampsia, similar estimations have become of great practical value in treatment. They have enabled the authors to give a more accurate prognosis and to anticipate by treatment the onset of grave complications. Should the uric acid content continue to rise and the carbon dioxide combining power of the blood appear to decrease in spite of treatment, they resort to delivery, and by this means they have been able to lower their mortality figures. They have come to the conclusion that blood chemistry gives a true index of the severity of the disease and of specific treatment needed. They found that the non-protein content of the blood remains within normal limits, except later in the disease, when a rise indicates an involvement of the kidneys. The blood uric acid is increased in eclampsia and preeclampsia, indicating a disturbance in its destruction in the liver. The uric acid contained in the blood may be regarded as a fairly safe criterion of the severity of the disease. The blood sugar level is not greatly disturbed. The alkali reserve is often greatly decreased, even to the level of a true acidosis. The carbon dioxide combining power is the best available index of the necessity of ante-acidosis treatment. The blood chlorides are markedly decreased, except in an occasional patient with œdema. Blood chlorides, blood theoneine and glutathione are within normal limits.

The Fœtal Mortality in Different Types of Toxæmia.

A. J. B. TILLMAN AND B. P. WATSON (*American Journal of Obstetrics and Gynecology*, January, 1935) discuss the fœtal mortality in all toxæmias of pregnancy in 1,036 cases. In the mild hypertensive group, that is, those with a systolic blood pressure of 130 to 150 and a diastolic pressure of 90 to 110 millimetres of mercury, of which there were 330 cases, the death rate was 3%. Those with a moderate degree of hypertension had a death rate of 3·6, whereas in the severe grade it was 30%; when associated with late albuminuria, it rose to 30·3%. In the nephritic group the death rate varied from 15·7% in the mild to 69% in the severe group. It was 24% in the preeclamptic and 41% in the eclamptic group. The authors have defined each group in detail, in order that confusion with other classifications will not arise.

Myocardial Degeneration with Pre-Eclamptic Toxæmia.

H. WINKLER (*Monatsschrift für Geburtshülfe und Gynäkologie*, December, 1934) discusses the clinical types of myocardial insufficiency seen in preeclamptic toxæmia. The more common one is where the heart is not damaged and possesses considerable reserve power. In such cases the blood pressure is generally raised to high levels owing to cardiac hypertrophy. In the other form the toxæmia has acted more directly on the myocardium, with consequent clinical signs of decompensation and alterations in electrocardiograms. Cloudy swelling of the myocardium is found at autopsy. The pulse rate is generally fast and is of poor volume.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Association Rooms, Adelaide Street, Brisbane, on April 5, 1935, DR. W. N. ROBERTSON, the President, in the chair.

The Causes of Death in Hospital Practice.

DR. J. V. DUHIG read a paper entitled "The Causes of Death in Hospital Practice: An Analysis of 380 Hospital Autopsies" (see page 647).

DR. M. GRAHAM SUTTON expressed the opinion that if Dr. Duhig's paper had done nothing more than stimulate interest in accurate clinical diagnosis he would be amply rewarded for the care he had taken. Dr. Sutton was quite sure that diagnosis was the most important single practical point for the medical practitioner, be he surgeon or physician. It was certainly disconcerting to find that diagnosis was inaccurate in such a large percentage of cases. Practitioners should set themselves a better standard, including methods of investigation; a medical school might stimulate and sharpen their clinical sense. It was very interesting to hear what an important part disorders of the circulatory system took in pathological processes, and when one considered the enormous amount of energy latent in this system, Dr. Sutton was not surprised at the results that occurred when there was any impediment to the circulation. With regard to the diagnosis of pyonephroses and the large number of cases of this disease, most of these cases were not really properly investigated, as the patients were too ill. In its earlier stages the condition must be accompanied by pyuria and some symptoms, and functional tests should be done on these patients. With regard to its ætiology, the condition occurred in children as well as adults, and it might possibly arise first as a hydronephrosis. There was something to be said for the theory of obstruction to the outlet of the bladder, as a lesion in that situation occurred in children, or for obstruction by an enlarged prostate *et cetera*. Hydronephrotic changes in the upper part of the urinary tract revolved round the fact that the *sphincter vesicæ* was opened by the contraction of the trigone muscle, which was inserted through the circular sphincter down to the verumontanum. The ureter was the only tube in the body with three muscular layers—an outer circular, a middle longitudinal, and an inner circular layer. Any obstruction or loss of elasticity in the position of the internal *sphincter vesicæ* altered this muscular control and caused hypertrophy of the trigonal muscle and longitudinal muscle bundles in the ureter and the first signs of hydronephrosis. There was stasis on top of this, then infection, and so everything necessary for a pyonephrosis. Dr. Sutton thanked Dr. Duhig for his interesting paper.

DR. CLIVE SIPPE thanked Dr. Duhig for his paper. There was one point to which he would like to draw attention: Dr. Duhig recorded four deaths from pernicious anæmia. In an English survey the age group from twenty-five to fifty years in 1928 showed a drop to 50% of the previous death rate. This was probably due to the introduction of liver therapy. But instead of a further drop, as one would have expected, there had been a rise. The probability was that patients, feeling well, had ceased taking liver or were taking less. It was important to remember that, once a diagnosis of pernicious anæmia had been made, treatment had to be kept up continuously.

DR. ALEX. MURPHY said that there was one factor operating which might explain the seeming failure to establish a correct diagnosis in so many instances, and that was omission to correct the original provisional diagnosis made and written on the history sheet by the resident medical officer who saw the patient on his admission. Although subsequent investigation often proved it to be erroneous, yet it frequently remained uncorrected until the case sheets came to the registrars. Thus the diagnoses on which Dr. Duhig had had to base his comparison often did not represent the final clinical opinion. One point he would like to mention: one patient had died of hydatid disease. Dr. Murphy had had a few patients suffering from this disease, but he had never been sure that the disease arose in Queensland; all the patients had admitted that they had lived in New South Wales or Victoria at some time in their lives. He had discussed this with his colleagues and found that they had had the same experience. He was not prepared to say that the disease never arose in Queensland, but he had never been able to satisfy himself that this was so.

There were five patients who had died of carcinoma of the stomach found at autopsy. Dr. Murphy did not agree with Dr. Duhig's remarks on this, as he thought the number probably depended on the fact that one could not always get permission from the relatives for a *post mortem* examination.

DR. NEVILLE G. SUTTON said it had been a pleasure and a privilege to listen to Dr. Duhig's paper. They all knew his enthusiasm and encouragement of this work at the Brisbane Hospital, and it was through his energy that the number of *post mortem* examinations had been so increased. A *post mortem* examination was one of the most important things in scientific medicine, and its results could be very salutary.

DR. N. W. MARKWELL thanked Dr. Duhig for his very estimable paper; he had attacked the subject very well. Dr. Markwell inquired regarding the ætiology of the seven cases of aortic regurgitation. Were they specific or otherwise?

DR. E. S. MEYERS congratulated Dr. Duhig and thanked him for a very pleasant evening; he liked his style of presentation and the way he used words, and considered that his remarks on death were very much to the point. While listening to the paper, the name of Mackenzie had run through Dr. Meyers's mind: "How difficult is the interpretation of symptoms." He considered that perhaps the results obtained were not too bad. There was plenty of work for future medical men. One was apt to decry these special methods; for example, in hydronephrosis in infants there might be a stricture in the urethra; to discover this was very difficult, and more men working longer hours would be required to find out these things. Until a short time ago at the Brisbane Hospital there had been no urologists, and the surgeons had done occasional operations. Now there was a definite urological department and ten or twelve operations were listed every week. The subject of thrombosis and embolism had been discussed a lot, but had not got much further forward. Willy Meyer, the great chest surgeon, in the *Surgical Clinics of North America* had said that one factor was stasis due to the position of the patient being prone and the venous circulation being slowed down. He now made a practice of elevating the foot of the bed. Dr. Meyers now followed this practice, and he recommended the article for perusal. With regard to the remarks on eclampsia, was Dr. Duhig under the impression that it was an adenoma or some pressure in the *sella turcica* which caused the condition? In those eclamptics who had a high blood pressure that fell suddenly, would he suggest that this sudden fall in pressure was due to the release of pressure in the *sella turcica*? He disagreed with Dr. Duhig on one point: he considered that the best use of the dead body was that it should go to the anatomist, not to the pathologist.

DR. S. F. MCDONALD said he had listened to the paper with pleasure. At the Hospital for Sick Children the sentimental factor was more important, and the percentage of *post mortem* examinations was much lower, but the staff was able to get much instruction from the work done there. This applied particularly to influenzal meningitis cases, which were fairly common in children. In frequency this condition took the place of the tuberculous meningitis seen in children in the south.

Dr. McDonald remarked that cases of abscess of the lung and respiratory passages following removal of tonsils and other operation in adults used to be fairly common, and the question always arose as to who was responsible. The

surgeon generally said they were embolic, while the anæsthetist said they were due to hæmorrhages into the air passages.

All children for removal of tonsils at the Hospital for Sick Children were now sent into hospital and great care was taken to prevent blood inhalation. In the last twelve months there had been no cases of post-operative emboli.

Dr. McDonald thought that embolism was more common where operation involved the veins in the posterior abdominal wall, those draining into the inferior *vena cava* rather than into the portal system.

DR. W. N. ROBERTSON spoke of post-operative emboli, which were so distressing and were apparently unpreventible. In his practice in ear, nose and throat work he had never had a case and would like to know the reason for them.

Dr. Duhig, in reply, thanked the speakers for their remarks. No indictment whatever was meant; the material had to be worked up, and he thought friendly criticism was one of the best forms of education. It was very difficult to assess the precise significance of these figures; the reasons for the *post mortem* examinations were given, and the conditions under which they were done were described.

With regard to the diagnosis of pyonephrosis, Dr. Sutton had shown, and Dr. Duhig had attempted to show, the condition of the patient on his admission to hospital. The condition could be diagnosed if one passed a ureteral catheter, but the patient was too ill to be disturbed like that. The question was how to stop him getting into that state. The subjects at *post mortem* examination were all men, and he thought that to find the possible causation for this dreadful condition, which removed men in the prime of life, some approach must be made during youth. The condition was probably congenital, and the patient had become accustomed to it from the very early stages. The man was not accustomed to be in good health and would not notice the condition; therefore the man must be found and treated early.

Dr. Duhig agreed with Dr. Sippe as to the deaths from pernicious anæmia, and that these people felt too well and stopped taking liver. They probably reached the aplastic stage finally and nothing would improve them. Four deaths from pernicious anæmia looked bad on the records. Dr. Duhig agreed with Dr. Murphy that the hospital was very understaffed, and he hoped that things would be better later on. They had a good institution and all worked well together and were eager to do good work. Men went out of their way to run special departments. The aortic regurgitation findings mentioned by Dr. Markwell were probably non-specific. Specific disease of the aorta was listed separately, but he constantly found in old people an atheromatous condition of two or three valves of the aorta with a bad closure. The thickening caused rigidity of the aortic tube, this dilated with the pressure of the blood, and there was a vicious circle. The whole thing pulled away, there would be a little chink and the aorta never closed; these cases, however, were always well compensated and probably there were no physical signs, or at any rate they would be very slight and easily overlooked. *Post mortem* they were found only by the water test, which was fairly severe.

In reply to Dr. Meyers, Dr. Duhig said that 41% of diagnoses were accurate and he was quite certain that if all patients who died had come to *post mortem* examination the percentage would have been much higher. The examinations were made because of the difficulty in diagnosis. Even so, Dr. Duhig thought there was still room for improvement, and this could come about only by more facilities for research and investigation. With regard to eclampsia, Cushing stated fairly dogmatically in the *American Journal of Pathology* that a basophile infiltration of the posterior lobe of the pituitary gland and of part of the media would be found in eclampsia. He produced a lot of clinical evidence, but Dr. Duhig had not been able to confirm it. Cushing's theory had not much to do with the bony bed of the gland, but probably more with the hormonal effect of the gland. If so, the fall in pressure would be comparable to the change-over in the menstrual cycle which would occur in pituitary lesions, so there might be something of this nature. Dr. Duhig's evidence went to show that this was not the only factor.

Dr. Duhig agreed with Dr. McDonald that there were not nearly the same number of *post mortem* examinations at the Hospital for Sick Children on account of sentimentality; this, however, was only a phase in emotional evolution and would pass. Dr. Duhig had asked Dr. Alex. Patterson what was being done in the pathology of children in England. He himself considered it a very difficult subject. All the lung abscesses found at *post mortem* examination had been due to teeth extraction under anæsthesia. None had been due to the removal of tonsils or operations on the respiratory passages.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Royal Alexandra Hospital for Children on April 11, 1935. The meeting took the form of a series of clinical demonstrations by the members of the honorary staff.

Disorder of Fat Metabolism.

DR. E. H. M. STEPHEN showed a boy, aged ten years, who was suffering from a disorder of fat metabolism. The boy had suffered from pneumonia and measles at six years of age. He began to lose weight early in 1934. Since November of that year he had had an oily, offensive discharge *per rectum*. This discharge stained his clothes and persisted up till the time of his admission to hospital. He had recurrences of diarrhœa with pain in the upper part of the abdomen.

He was admitted to hospital on February 26, 1935, with a normal temperature and normal pulse rate. There was no marked pallor or icterus and he was not wasted. There was generalized soft distension of the abdomen, but no tenderness. No tumours could be felt. The liver was of normal dimensions. The motions were slate-coloured, copious and fatty. Their odour was offensive and cheesy. Though there was a faintly positive reaction to the Mantoux test, the von Pirquet test gave no reaction. No tubercle bacilli were found on examination of the fæces. X ray examination yielded no evidence of pulmonary tuberculosis. The Wassermann test gave no reaction. An analysis of the stools did not conform to that of cœliac disease. It was concluded that there was a disorder of pancreatic function of very unusual type and probably of a temporary nature.

Analysis of dried stools showed: total fat, 52% (normal 25%); unsplit fat, 38% (normal 25%); split fat, 62% (normal 75%).

Microscopic examination revealed soap plaques and fat-free globules in the fæces. There was no occult blood. Numbers of muscle fibres exhibiting a striated appearance were present in the fæces, suggesting interference with the function of the pancreas. On the other hand, the fact that the diastatic index was normal did not add support to this. The blood cholesterol was 80 milligrammes per 100 cubic centimetres of serum, which was consistent with such disorders. The sedimentation rate was normal. The blood sugar at 3 p.m. was 100 milligrammes per 100 cubic centimetres of blood. The lævulose test revealed no disturbance of liver function.

After treatment with a fat-free diet the motions, though not normal, were improved in character. The total fat was 13%, the unsplit fat was 17%, the split fat was 83%. The patient appeared well and energetic, and there was no further oily discharge.

Diabetes Mellitus.

Dr. Stephen also showed a boy, aged eleven years. The patient was shown to illustrate the management of a case of diabetes. His weight was 29·25 kilograms (65 pounds). The maximum caloric requirements of a boy of his age, living an active life, were 2,515 calories. This patient was in hospital, but ambulatory, and was allowed 1,872 calories. He was given: protein, 72·3 grammes (289 calories); fat, 112·2 grammes (1,009 calories); carbohydrate, 143·5 grammes (574 calories).

An endeavour was being made to increase the proportion of carbohydrate and to lower that of fat. He was receiving

twenty units of insulin in the morning and ten units in the evening. The results of urine tests were not yet satisfactory, as reduction of Benedict's solution was still taking place, though ketone bodies were seldom present.

Anterior Poliomyelitis.

Dr. Stephen's third patient was a girl, aged nine years, who was admitted to hospital on March 18, 1935, with a history of fever, headache and vomiting for three days, with a weakness of the arms, noticed on the day of admission.

At the time of the patient's admission to hospital the cerebro-spinal fluid was under moderate pressure. The cells numbered 105 per cubic millimetre; 80% were lymphocytes and 20% nuetrophile cells. The chloride content was 705 milligrammes *per centum*. The tests for globulin and glucose both gave positive results.

Dr. Stephen pointed out that the patient was showing a flaccid paralysis with wasting of the muscles of the shoulder girdle and of the flexors and extensors of the forearm on both sides. She was equipped with an aeroplane splint and was undergoing muscle reeducation.

Pseudohypertrophic Muscular Dystrophy.

Dr. Stephen showed three patients who were suffering from pseudo-hypertrophic muscular dystrophy. They had wasting of the shoulder girdle muscles, of the *pectoralis major*, thigh muscles and *latissimus dorsi* muscles, with pseudohypertrophy of the calf muscles, infraspinati and gluteal muscles. They also showed lordosis and characteristic waddling gait.

One patient had wasting of the external oblique muscle of the abdominal wall. In the most advanced case the patient was unable to rise from the recumbent position by climbing up himself.

Epilepsy.

DR. T. Y. NELSON demonstrated a case of epilepsy in a child whose condition had been relieved by operation.

At the age of six weeks the child had sustained a head injury as the result of a motor accident. No serious symptoms were noticed at the time, but at the age of eighteen months the child commenced to have attacks of *petit mal*. These were followed by Jacksonian fits affecting the left arm. The number of fits had increased, had become major in character, and at the time of admission to hospital the child had been having as many as five or six in the day.

Encephalography revealed absence of cortical markings on the right side. Operation revealed an area of adhesions between the *dura mater* and the cortex in the right parietal region. The adhesions were divided and the dura was replaced. Operation was followed by a period of complete cessation of fits for three months and the child had improved in general health. At the time of the meeting, however, attacks of *petit mal* had been observed again.

Osteomyelitis of the Pelvis.

Dr. Nelson then demonstrated three cases of osteomyelitis affecting the pelvic bones, which illustrated the variation in virulence of the infecting organisms.

The first child, a boy of twelve, was admitted to hospital with a large swelling on the left side, extending above the inguinal ligament almost to the costal margin. There was limitation of movement at the hip joint and pyrexia and leucocytosis were present. A large collection of pus was evacuated extraperitoneally above the inguinal ligament and the cavity was drained. After months of treatment, during which time the cavity was irrigated by the Carrel-Dakin method and a blood transfusion was given, the sinus eventually healed. The final result after eighteen months showed a hip joint ankylosed in abduction.

The other two cases of osteomyelitis affected the descending ramus of the pubis and the ischium. The children had similar clinical signs, consisting of acute onset of pain, rise of temperature and tenderness in the perineum. In both cases pus was evacuated in the upper part of the thigh by an incision extending along the line of the adductor muscles. After evacuation of the pus the cavity was packed with vaseline gauze, which was left undisturbed for three weeks. After this time the wound rapidly healed and the patients were discharged from hospital within six weeks of the operation.

Supracondylar Fracture of the Humerus.

Dr. Nelson's last case illustrated the use of skeletal traction in certain types of supracondylar fracture of the humerus. The patient was a girl of six who showed the comparatively rare displacement anteriorly of the lower fragment. It was pointed out that in these cases the flexed position of the forearm would not retain the fracture in position and skeletal traction was indicated. A Kirschner wire had been drilled through the olecranon and extension of three pounds applied in the line of the humerus had brought the fragments into good position.

Congenital Ectodermal Dysplasia.

DR. LORIMER DODS showed a patient who was suffering from congenital ectodermal dysplasia. This case will be reported in full in a subsequent issue.

Large Ulcer of the Groin.

DR. P. L. HIPSLEY showed a boy, aged ten years, who was admitted to hospital on April 27, 1934, suffering from an ulcer which had been present in the right groin for two years. The condition had started as a lump in the groin following a cut in the foot. The patient was treated for twenty-one months at another hospital and was given two applications of deep X ray therapy, the first in June, 1933, and the second in February, 1934. Since February, 1934, the ulcer had definitely increased in size.

On May 24, 1934, treatment by the constant drip, Carrel-Dakin method, was adopted. The limb was put on extension to overcome a flexion deformity at the hip. The extension caused severe pain and had to be discontinued. Seven "Novarsenobenzol" injections were given.

On September 28, 1934, an antivirus dressing was applied and a blood transfusion was given.

On December 14, 1934, intravenous injections of antimony potassium tartrate were started. Definite improvement occurred. The patient had had eight injections and the ulcer was being dressed twice a day with pads soaked in Dakin's solution.

Dr. Hipsley pointed out that the Wassermann test had given no reaction and that attempts had been made to culture organisms from the blood, but without success. Numerous smears had been taken from the ulcer at varying intervals. Cocci had been found in pairs and occasionally in short chains. *Staphylococcus aureus* and coliform bacilli had been cultured. A biopsy was taken and a report of chronic inflammatory tissue was received.

Tuberculous Adenitis of the Groin.

DR. F. C. ROGERS showed a boy, aged eight years, who was admitted to hospital on November 14, 1934. The child had come from Italy eight months previously and there was no history of tuberculosis in the family. On admission the history was that an abscess in the groin had been incised in the out-patient department two months previously and that the wound had continued to discharge. A sinus was present in the groin; it discharged pus and the skin around the opening of the sinus was of a bluish colour. On examination the heart, lungs and central nervous system appeared to be normal. The tonsils were enlarged. The femur and hip joint were examined by X rays and no abnormality was discovered. No reaction was obtained to either the Wassermann or the Kahn test. On examination of the blood-stained discharge from the sinus pus cells and Gram-positive diplococci were found in the smear, and hæmolytic streptococci were obtained on culture. On examination of the excised glands miliary tubercles were found and tubercle bacilli were present. Reactions were obtained to the von Pirquet and the Mantoux tests. After the glands were excised the limb was put on an extension apparatus and healing occurred slowly.

Congenital Deformity of the Spine.

Dr. Rogers also showed a girl, aged twelve years, who had been treated at the age of eighteen months for what was thought to be rheumatism in the legs. The legs appeared to be paralysed, but the child made a good recovery. *Pes cavus* appeared insidiously during the following years. At the age of eleven the child attended a physical culture class and soon afterwards a lump was noticed in her back. She also suffered from pneumonia.

The child was admitted to hospital on February 12, 1934. At this time the lump had been present in the back for four months. She had lost no weight, had had no night sweats and no night starts. The lump was painless. The patient was a sallow-complexioned girl who lay comfortably in bed. The spinal deformity was situated in the lower thoracic region. No abnormality was detected on examination of the lungs, heart, central nervous system, throat and urine.

On February 13, 1934, an X ray examination of the spine was made. The radiologist reported the presence of a tuberculous lesion involving the tenth, eleventh and twelfth dorsal vertebræ; the body of the eleventh vertebra was thought to be collapsed.

On March 15, 1934, the radiologist reported that the appearance suggested a "congenital variation hemivertebra wedge-shaped in lateral view". The lamina of the vertebra on the right side had not developed. The development of the neural arch in the sacral region was defective. No evidence of tuberculosis was seen. Both the von Pirquet and Mantoux tests were applied, but no reaction was obtained.

Later the child was sent to the convalescent branch of the hospital at Collaroy. Subsequently operations were carried out for correction of the deformity of the foot.

Bronchoscopy.

Metallic Collar Stud in a Lower Lobe Bronchus.

DR. N. H. MEACLE showed a boy, aged eleven years, who was admitted to the Royal Alexandra Hospital for Children on December 1, 1934, with the history of having "swallowed" a small back collar stud six days previously. On examination of the patient, a well nourished boy, harsh breath sounds could be heard in the upper half of the left side of the chest. X ray examination revealed a collar stud near the hilum of the left lung.

On December 4, 1934, under ether anæsthesia, a five millimetre Jackson's bronchoscope was passed as far as the left lower lobe bronchus. No foreign body was detected. A few days later the patient developed scattered rhonchi in both lungs, and by December 11, 1934, was running a high temperature. Chest examination on that date revealed an impaired percussion note at the left base, with diminished breath sounds. There was also a patch of tubular breath sounds at the medial border of the left scapula, just above the inferior angle. X ray examination of the chest indicated the foreign body to be in the region of the left lower lobe bronchus, also diminished aeration and collapse of the left lower lobe. The contralateral lung showed compensatory emphysema. The patient continued to run a hectic chart, progressively lost weight, and had occasional attacks of vomiting and breathlessness. Examination on December 28, 1934, revealed absolute dulness at the base of the left lung posteriorly, with absence of breath sounds and of vocal resonance. X ray examination of the chest showed that the dulness at the left base was increasing. There appeared to be massive collapse of the left lower lobe.

On December 31, 1934, a five millimetre Jackson bronchoscope was again passed into the left lower lobe bronchus as far as possible, but the stud could not be seen. Muco-pus was aspirated from the bronchus. During the following week the patient's condition improved and the temperature was less elevated.

On January 8, 1935, the bronchoscope was again passed as far as possible into the left lower lobe bronchus and an X ray picture was taken with the bronchoscopic tube *in situ*. The stud was shown to be about half an inch beyond the tip of the bronchoscopic tube. The stud was then felt for and grasped by the foreign body forceps and removed. The patient, whose condition after the operation was described as moderately good, was placed in a steam tent for the following twenty-four hours. After removal of the foreign body the temperature dropped to normal and remained normal, and during the next five weeks the clinical signs in the left side of the chest gradually returned to normal and the patient was discharged from hospital in excellent health.

Peanut in the Right Bronchus.

Dr. Meacle also showed a child, aged two years, who was admitted to the Royal Alexandra Hospital for Children on December 28, 1934, with the history of having on that day commenced to cry while eating a peanut and after which he was noticed to wheeze.

On examination the child cried lustily and did not appear to be distressed. The chest was resonant throughout and breath sounds were vesicular. X ray examination on December 29, 1934, revealed the right lung to be unusually emphysematous, due probably to a transradiant foreign body. A seven millimetre bronchoscope with proximal illumination was passed in the right bronchus and half a peanut kernel was seen. Grasping the peanut with the foreign body forceps in an attempt to remove it in one piece caused the nut to break up into small pieces so that only one piece of the foreign body was removed at the first session.

On December 31, 1934, X ray examination revealed consolidation of the base of the right lung, due to blocking of the right lower lobe bronchus by a piece of peanut. Bronchoscopy was carried out at intervals of about a week on four occasions until all the peanut had been removed. After the last piece of nut had been removed from the right lower lobe bronchus, the lung quickly returned to normal and the child was discharged from hospital quite well on February 15, 1935.

Bathurst Burr in the Right Main Bronchus.

Dr. Meacle's last patient was a girl aged five years, who was admitted to the Royal Alexandra Hospital for Children on February 22, 1935, with the vague history of having "swallowed" a grasshopper three weeks previously. Cough and occasional distressed breathing had been present ever since. On examination the chest appeared to move evenly with good excursion. Palpation appeared normal. The percussion note was found to be resonant over all areas, and the breath sounds were harsh, vesicular and much reduced in intensity over the right side. Coarse and musical rhonchi were present in the right interscapular region. Other systems were clear and there was no pyrexia. X ray examination revealed the right lung to be markedly emphysematous, suggesting the presence of a transradiant foreign body in the right bronchial tube.

On February 26, 1935, under ether anæsthesia, a five millimetre Jackson's bronchoscope was passed and the Bathurst burr was located in the right main bronchus. The burr was closely surrounded by and incorporated in gross inflammatory reaction. The foreign body was removed with forceps and the child returned to the ward in good condition and was nursed in a steam tent for twelve hours. Recovery was uninterrupted. On February 28, 1935, X ray examination showed the lungs to be quite normal and breath sounds were clear. On March 6, 1935, the child appeared to be quite well and was discharged from hospital.

Cœliac Disease.

DR. MARGARET HARPER showed two patients who were suffering from cœliac disease.

The first patient was a girl, aged two years and three months. She was admitted to hospital on October 16, 1934. She was an only child; there was no history of tuberculosis in the family.

The history was that the child had been ill for three months. She vomited once a day and had had diarrhœa for three months; she passed five or six green motions every day. The abdomen had been enlarged for two weeks; for several months the child had been losing weight.

The fat content of the stool was estimated; the total fat was 54%, unsplit fat was 31%, and split fat 69%.

The glucose tolerance test was performed. The fasting specimen contained 84 milligrammes *per centum*; the figures half an hour, one hour, one and a half, and two hours after the administration of glucose were respectively 95·5, 87·5, 75·0 and 80·0 milligrammes.

The erythrocytes numbered 4,520,000 per cubic millimetre, the hæmoglobin value was 46%, the leucocytes numbered 9,400 per cubic millimetre.

On X ray examination a generalized transradiancy, suggestive of some nutritional dystrophy, was found.

Treatment consisted in the administration of a high protein diet, in the giving of irradiated ergosterol and iron. On this treatment the child gained weight and was discharged from hospital on December 10, 1934.

The second patient was a boy, aged five years, who was admitted to hospital on February 5, 1935.

The patient was the fourth of seven children; all the other children were healthy. The patient's only other illness was pertussis.

The history of the present illness was that the child was fed on cow's milk till the age of fourteen months and that up to this stage its progress had been satisfactory. It then refused food and became very thin, having three to four loose motions every day. The abdomen had been increasing in size for three to four years. For three months prior to the child's admission to hospital it had vomited occasionally. At the time of its admission to hospital examination revealed a thin, pale child, with wasted buttocks and a distended abdomen.

Dr. Harper pointed out that numerous pathological and biochemical investigations had been carried out. The von Pirquet test yielded no reaction. A test meal revealed slow secretion of hydrochloric acid.

The glucose tolerance test was carried out. Before the administration of glucose the blood sugar content was 96 milligrammes *per centum*. The figures half an hour, one hour, one and a half hours, and two hours after the administration of glucose were respectively 105, 120, 100 and 92 milligrammes.

The fat content of the stool was estimated; the total fat was 48%; unsplit fat was 22%, split fat was 78%.

The blood phosphorus was 4·7, the blood calcium was 11·2 and the blood cholesterol was 70 milligrammes *per centum*. The Wassermann test gave no reaction.

On February 7, 1935, the erythrocytes numbered 3,110,000 per cubic millimetre, the hæmoglobin value was 59%, the leucocytes numbered 7,000 per cubic millimetre.

On March 26, 1935, the erythrocytes numbered 4,230,000 per cubic millimetre, the hæmoglobin value was 55%, the leucocytes numbered 8,500 per cubic millimetre.

X ray examination of the long bones revealed multiple lines of arrested growth, and depletion of the calcium content—appearances that were consistent with a diagnosis of cœliac disease.

Treatment consisted in the administration of iron, calciferol and a fat-free diet. The child was gaining weight.

Pituitary Tumour.

DR. L. H. HUGHES showed a girl who was thought possibly to be suffering from a pituitary tumour. The child was always languid and tired and was very irritable. For six weeks she had complained of severe headaches and of pain in the neck. Her tonsils and adenoids had been removed three months previously and since then she had put on weight. She had not vomited. The sugar tolerance was normal. Double papillœdema was found to be present on examination of the eyes. Neither the Kahn nor the Wassermann test yielded a reaction. On X ray examination the *sella turcica* was seen to be shallow and there was slight enlargement in its anterior part, suggesting slight enlargement of the anterior part of the pituitary gland.

Glandular Disease.

DR. M. J. PLOMLEY showed a boy, aged seven years, who was admitted to hospital on April 8, 1935. Before the present illness commenced the child had suffered from pertussis, measles, mumps and laryngeal diphtheria. His tonsils and adenoids had been removed.

The history of the present illness was that the boy was apparently well until four weeks before his admission to hospital, when his mother noticed some swelling in the glands in his neck. He complained of generalized abdominal pain, particularly after taking exercise; the pain was in no way related to food. For one week he had a "brassy" cough which became more constant and rapidly more severe. For one week he had dyspnœa, which rapidly became worse. He had anorexia, but no vomiting. He had sweated considerably for one month and was becoming paler.

At the time of his admission to hospital the patient had a pyrexia of 37·8° to 38·3° C. (100° to 101° F.). Discrete glandular swellings were present in both sides of the neck. Similar swellings were present in the groins, where palpation was painful. The spleen was not palpable. On the left side of the chest the breath sounds were harsh and vesicular. They were diminished in the subapical region, where they were broncho-vesicular; they were inaudible at the left base. Vocal resonance was diminished at the left base, where ægophony was present. The percussion note became progressively more dull from apex to base. On the right side of the chest the breath sounds were harsh and vesicular, with occasional rhonchi. The percussion note was impaired. Examination of the chest stimulated attacks of coughing and fatigued the child.

The abdomen was tender in all areas; no tumours were palpable.

The central nervous system and the urine were clear.

A blood count yielded the following information:

Erythrocytes, per cubic millimetre ..	2,610,000
Hæmoglobin value	40%
Colour index	0·76
Leucocytes, per cubic millimetre	1,800
Polymorphonuclear neutrophile cells	15%
Lymphocytes	78%
Monocytes	7%
Eosinophile cells	—

No abnormal red or white cells were seen.

X ray examination of the chest revealed a large opaque mass on the left side of the chest, extending into the right hilar region.

Dr. Plomley also showed a boy, aged nineteen months, who was admitted to hospital on April 3, 1935. The child had previously been healthy. He had been ill for four days with a swelling in the right side of the neck; he was drowsy, but had not vomited. His shins were swollen and red for two days. He was feverish and constipated. He had no urinary symptoms.

Examination at the time of the boy's admission to hospital revealed a semi-discrete mass of glands in the right side of the neck, firm in consistency, but not obviously painful. He had bilateral erythematous swellings of both shins, painful to palpation. Other glands were just palpable. The fauces were congested. The temperature was 37·2° C. (99° F.), the pulse rate was 120 and the respirations numbered 28 in the minute. Other systems were clear.

A blood count revealed the following information:

Erythrocytes, per cubic millimetre ..	4,140,000
Hæmoglobin value	53%
Colour index	0·62
Leucocytes, per cubic millimetre	8,000
Neutrophile cells	53%
Lymphocytes	29%
Monocytes	7%
Eosinophile cells	11%

The von Pirquet test yielded no reaction. The urine was normal.

Dr. Plomley said that the glands in the neck and the swellings in the legs had subsided considerably under the influence of fomentations and with the administration of potassium and iron. The temperature had remained between 37·2° C. (99° F.) and normal. The child had become less fretful.

Volkmann's Ischæmic Paralysis.

DR. W. VICKERS showed two patients who were suffering from Volkmann's ischæmic paralysis. The first, a girl, aged seven years, had fractured her right arm above the elbow seven weeks before her admission to hospital. On her admission to hospital she had a right claw hand, flexion of the elbow was restricted, the muscles of the forearm were wasted and movements of the wrist were restricted. X ray examination revealed backward displacement of the lower fragment.

Treatment had consisted in manipulation under anæsthesia, osteotomy of the radius and ulna, and manipulation of the forearm.

The second patient was a boy, aged seven years, who had fractured his right humerus three months prior to his admission to the Royal Alexandra Hospital. He had been discharged from a country hospital with contracted fingers.

On his admission to the Royal Alexandra Hospital he had flexion contraction of the fingers and considerable wasting of the extensors and flexors of the forearm. X ray examination revealed considerable backward displacement of the lower humeral fragment, with slight medial displacement and rotation.

Treatment had consisted in: (i) dissection of the ulnar nerve so that it was freed from involvement, (ii) osteotomy of the lower end of the ulna and of the upper end of the radius, (iii) the application of plaster bandages, (iv) massage.

Penetrating Injury to the Eye.

DR. N. M. GREGG showed a girl, aged eleven and a half years, who was admitted to hospital at 9 p.m. on February 18, 1935, with a fish hook in her right eye. The hook had been in the eye since 5 p.m. on that day.

On examination the fish hook was found penetrating the cornea in the four o'clock position. Operation was performed one hour after the child's admission. The fish hook was removed by pushing the barb to the surface and breaking it off; a small piece of iris was removed. Intramuscular injections of sterile milk, four in number, were given, extending over six days. Atropine drops were instilled and atropine ointment was used.

Acute Mastoiditis with Streptococcal Meningitis.

DR. RAMSAY BEAVIS showed a boy, aged eleven years, who was admitted to hospital on February 6, 1935, feverish and vomiting, and complaining of severe headache. He had had a discharging left ear for two weeks.

On examination the left ear was discharging freely, no swelling or tenderness was present, neck rigidity was present, the patient's back was stiff, and a double Kernig's sign was elicited. Lumbar puncture yielded turbid cerebrospinal fluid, from which streptococci were grown. A mastoid operation was performed on the day of admission. The lateral sinus was exposed; the middle cranial fossa and the cerebellum were exposed and the *dura mater* was incised. The patient recovered completely.

Skiagrams.

DR. H. R. SEAR showed several skiagrams.

The long bones of a child showed well established lead poisoning, with the characteristic opaque bands at the end of the diaphyses. Progress skiagrams were shown demonstrating the reduction in the density of this band and its altered situation with growth.

A second group included skiagrams of the skull, pelvis and some of the long bones of a patient affected with lipoid histiocytosis of the massive type. The changes in the tibia and in the pelvis were very similar to those of *osteitis fibrosa*, but the area in the skull was typical of xanthoma, suggesting that the lesion was lipoid histiocytosis. This was confirmed by biopsy (Dr. Tidswell).

Films of marble bones were shown, with clubbing of the ends and extraordinarily marked transverse opaque bands. Dr. Sear explained that transverse bands were not uncommon, but it was unusual to see them developed to such a degree as in these films.

Marble bones in an old case showed the clubbing, but with vertical striation. A macerated bone affected with osteopetrosis had been described as fluted like a Corinthian column, and one would expect this to give such longitudinal striation.

Dr. Sear also showed skiagrams of an extreme case of Ollier's disease; in this case the lesion was bilateral.

Intestinal Obstruction in Children.

DR. FRANK TIDSWELL demonstrated a number of pathological specimens illustrative of intestinal obstruction in children. These included: atresia of the œsophagus (two specimens); congenital hypertrophic stenosis of the pylorus, (*a*) untreated, (*b*) of four days' duration, and (*c*) four months after operation; congenital malformations of the duodenum, jejunum and ileum; obstruction due to tumour; volvulus; incarcerated intussusception; gangrene following intussusception; intussusception *post mortem*; Meckel's diverticulum, various sizes; neuromuscular dystrophy, (*a*) achalasia of the ileum, (*b*) Hirschsprung's disease, (*c*) multiple dilatations of the intestine.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Laurie, Elizabeth Frances Lois, M.B., B.S., 1933 (Univ. Sydney), Sydney Hospital, Sydney.

White, Harold Newbon, M.B., B.S., 1931 (Univ. Sydney), Royal Alexandra Hospital for Children, Camperdown.

Obituary.

RENZO ROSATI.

WE regret to announce the death of Dr. Renzo Rosati, which occurred on May 12, 1935, at Sydney, New South Wales.

JAMES PRESTON HOCKEN.

WE regret to announce the death of Dr. James Preston Hocken, which occurred on May 15, 1935, at West Wallsend, New South Wales.

SAMUEL HUBERT SECCOMBE.

WE regret to announce the death of Dr. Samuel Hubert Seccombe, which occurred on May 17, 1935, at Brisbane, Queensland.

THE GEORGE MACDONALD TESTIMONIAL FUND.

THE undermentioned subscriptions have been received for the George Macdonald Testimonial Fund:

£3 3s.: Dr. Marjory Little, Dr. N. D. Royle, Dr. C. B. Blackburn.

£2 2s.: Dr. B. T. Edye, Dr. Jessie S. Freeman, Dr. Colin M. Edwards.

£2: Dr. George Bell.

£1 1s.: Mr. W. A. Selle, Dr. R. J. Silverton, Dr. W. I. Hotten, Dr. M. R. Flynn, Dr. F. S. Cotton, Dr. Edward Rivett, Dr. M. A. Fletcher, Sir Mungo W. MacCallum, Professor C. E. Fawsitt, Dr. T. M. Greenaway, Dr. J. H. D. Edwards, Dr. G. Tahmindjis, Dr. F. C. B. McKay, Dr. J. T. Paton, Dr. F. G. Antill Pockley, Dr. D. W. Magill, Dr. Hugo de Burgh, Dr. W. C. Sawers, Dr. Cotter Harvey, Dr. H. R. Mallam, Dr. J. G. Edwards, Dr. C. J. Wiley, Dr. A. J. Cunningham, Dr. R. V. Graham.

11s.: Dr. Maynard Rennie.
10s. 6d.: Dr. N. H. Saxby, Miss Fosbery, Dr. K. G. Lawrance, Dr. A. W. D'Ombrain, Dr. R. E. Longworth, Dr. N. D. Barton, Dr. M. D. Harper, Dr. T. H. Small, Dr. Yorke E. Pittar, Dr. H. B. Cribb, Dr. H. G. Armstrong.
5s. 6d.: Dr. D. Reid.

AUSTRALIAN ARMY MEDICAL CORPS DINNER.

THE Committee of the Officers' Mess of the Australian Army Medical Corps, Second Military District, has arranged to hold the annual dinner at the Imperial Service Club, Barrack Street, Sydney, on Friday, May 31, 1935, at 7.15 p.m. A cordial invitation is extended to all those who served with the medical services during past campaigns. The inclusive cost of this dinner will be 15s. Those who intend to be present are asked to inform the Honorary Secretary, c.o. Victoria Barracks, Sydney, as soon as possible.

Books Received.

AMEBIASIS AND AMEBIC DYSENTERY, by Charles F. Craig, M.D., M.A., F.A.C.P., F.A.C.S.; 1934. London: Baillière, Tindall and Cox. Royal 8vo., pp. 315, with illustrations. Price: 22s. 6d. net.

Diary for the Month.

MAY 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAY 30.—South Australian Branch, B.M.A.: Branch.
MAY 30.—New South Wales Branch, B.M.A.: Branch.
MAY 31.—Queensland Branch, B.M.A.: Bancroft Memorial Lecture.
JUNE 4.—Tasmanian Branch, B.M.A.: Council.
JUNE 5.—Western Australian Branch, B.M.A.: Council.
JUNE 5.—Victorian Branch, B.M.A.: Branch.
JUNE 6.—South Australian Branch, B.M.A.: Council.
JUNE 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JUNE 11.—Tasmanian Branch, B.M.A.: Branch.
JUNE 14.—Queensland Branch, B.M.A.: Council.
JUNE 15.—Victorian Branch, B.M.A.: Branch.
JUNE 18.—Tasmanian Branch, B.M.A.: Council.
JUNE 18.—New South Wales Branch, B.M.A.: Ethics Committee.
JUNE 19.—Victorian Branch, B.M.A.: Clinical Meeting.
JUNE 19.—Western Australian Branch, B.M.A.: Branch.
JUNE 20.—New South Wales Branch, B.M.A.: Clinical Meeting.
JUNE 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments.

Dr. H. McI. Birch (B.M.A.) has been appointed Superintendent, Parkside Mental Hospital, and Superintendent of the Hospital for Criminal Mental Defectives, South Australia.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xiii, xiv, xv.

AUSTIN HOSPITAL FOR CANCER AND CHRONIC DISEASES, HEIDELBERG, VICTORIA: Junior Resident Medical Officer.
CHILDREN'S HOSPITAL (INCORPORATED), PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officer.
LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officers.
LIVERPOOL STATE HOSPITAL AND HOME, LIVERPOOL, NEW SOUTH WALES: Honorary Surgeon.
PUBLIC SERVICE BOARD, SYDNEY, NEW SOUTH WALES: Junior Resident Medical Officers.
REPATRIATION COMMISSION, PERTH, WESTERN AUSTRALIA: Medical Officer.
THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Junior Resident Medical Officers.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointment and those desiring to accept appointments to any COUNTRY HOSPITAL, are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Officer of Health, District Council of Elliston. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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